ARCHIVES OF OTOLARYNGOLOGY

VOLUME 13 JUNE, 1931 NUMBER 6

NEURO-OTOLOGICAL OBSERVATIONS IN CONCUSSION OF THE BRAIN*

FRED H. LINTHICUM, M.D.
AND
CARL W. RAND, M.D.
LOS ANGELES

Vertigo, in some form, is perhaps the most persistent symptom following concussion of the brain. Approximately 90 per cent of the patients describe some degree of equilibratory disturbance ranging from a mere feeling of uncertainty to actual systematized vertigo.

In work on industrial accidents it is frequently difficult to determine how much foundation in fact there is in the constant complaint of posttraumatic dizziness; in other words, it is difficult to evaluate how much is organic, and how much is functional. If a person who has sustained an injury to the head states that he is dizzy, what means has one to corroborate his statement? This question is especially important when the factor of compensation for disability must be considered. A means at hand to throw light on the problem seems to offer itself in the functional tests of the kinetic-static labyrinth, over the responses of which the patient has little, or no, control. This circumstance does not obtain in the functional tests of hearing in which the personal element plays no small part in interpretation. Abnormal neurologic observations are frequently absent, as is brought out in the appended reports of cases in which the patients suffered from concussion. If this is true, and the patient still complains of being dizzy, one cannot dismiss his claim as valueless, if it can be demonstrated objectively that the equilibratory apparatus is deranged. If, on the other hand, a person states that he is incapacitated for work on account of dizziness, and a normally functioning vestibulo-auditory mechanism can be demonstrated, it may be assumed that either there has been an exaggeration of the injured person's subjective symptoms, or there is actually present a vertigo of

* Submitted for publication, Nov. 28, 1930.

* Read in part as a candidate's thesis before the American Laryngological, Rhinological and Otological Society, Atlantic City, N. J., May 30, 1930, by Fred H. Linthicum, M.D.

psychogenic origin; whether or not the latter form does occur does not concern us at this time.

The series of concussional cases presently to be reported, when viewed purely from neuro-otological evidence, would seem to indicate that there is an actual basis for the symptom of dizziness, since, in no instance, have entirely normal vestibular responses been demonstrated and in the majority of cases the deviation from normal was sufficiently large to class them as being distinctly pathologic. One might therefore infer that an injustice has frequently been done by denying compensation for alleged incapacity to work on account of postconcussional dizziness. At least it would seem that the patient is entitled to an investigation of his equilibratory apparatus by functional tests of his inner ear.

The following observations have been made in an attempt to evaluate by objective evidence the relation of traumatic vestibular damage to subjective postconcussional vestibular symptoms. The material from which the data were compiled consists of thirty-six instances of concussion of the brain which were referred by the California State Compensation Insurance Fund, the Los Angeles General Hospital and through other channels. In a number of instances it has been possible to follow the patient by neurologic and neuro-otological observation over periods ranging from nine days to two years.

A general history, as well as a neurologic and a general physical examination, was made in each instance. Following this a history pertinent to symptoms of the inner ear was obtained and this was completed by the carrying out of functional hearing tests, and the observation of spontaneous and elicited vestibular phenomena.

HISTORICAL DATA

As far back as 1890, Risk[1] mentioned traumatic injury to the labyrinth. As was usual during that period, the labyrinth was considered only from the standpoint of its acoustic function. He made no comment on vestibular symptoms as considered today.

In 1901, Halsz[2] described definite vestibular symptoms such as the presence of postural vertigo as a result of a gunshot wound. He described two classes of trauma of the inner ear: direct traumatic injury and indirect injury brought about by sudden fluctuations of pressure in the external auditory canal, such as loud sounds or concussion. Five

1. Risk, E. J. E.: A Case of Traumatic Injury to the Labyrinth of the Right Ear, Successfully Treated with Injections of Pilocarpine, Brit. M. J. **1**:234, 1890.

2. Halsz, Heinrich: Zur Lehre der Labyrinthverletzung, Wien. med. Wchnschr. **51**:1538, 1901.

years later, Rhese,[3] in reporting a series of forty-five cases of people suffering from concussion of the brain, noted as frequent symptoms disturbances of equilibrium and diminished hearing of the receptor type. His observations at this date had to do mostly with spontaneous postconcussional symptoms of the inner ear as distinguished from induced phenomena. In 1905, Passow [4] made the observation that in severe injuries to the skull the inner ear is practically always involved. In 1909, Stenger [5] described in detail microscopic and gross pathologic observations in the inner ear following death from fracture of the skull and concussion. He concluded that the direct effects of labyrinthine concussion are: (1) alteration of the nerve, ganglion cells and nerve endings; (2) extravasations of blood, and (3) changes in pressure in the perilymph and endolymph content of the labyrinth.

In 1914, Emil Amberg,[6] in an article entitled "Injuries to the Head and Ear Disturbances," discussed the effects of indirect damage to the inner ear following injuries to the head. He viewed the problem, however, only from the standpoint of acoustic involvement. He mentioned "commotion of the acoustic nerve" caused by small extravasations of blood into the labyrinth. He noted that comparatively slight blows can cause severe derangement of the inner ear, and quoted Politzer to the effect that after such injuries connective tissue has been found in the labyrinth as early as the end of the fifth week. However, his conclusions were based on observations confined entirely to the acoustic portion of the inner ear.

In his first article which appeared in 1916, during the analysis of six cases of injury to the head, Mygind [7] made the observation that it was difficult to distinguish between organic and functional vestibular disease. Later, in 1917, in a review of a previous work, "Vestibular Examinations of Patients Suffering from Injuries to the Head," he analyzed the vestibular observations in 142 patients who had received injuries to the head; half of this number had suffered from concussion. A spontaneous nystagmus was observed in 26 instances; 44 of the patients had spontaneous past pointing. In our series the percentage of the

3. Rhese: Ueber die Beteiligung des inneren Ohres nach Kopferschütterungen mit vorzugweiser Berücksichtigung derjenigen Falle, bei denen die Hörfähigheit für die Sprache garnicht oder nur in einem praktisch nicht in Betracht kommenden Grade gelitten hat, Ztschr. f. Ohrenh. **52**:320, 1906.

4. Passow, K. A.: Die Verletzungen des Gehörorganes, Munich, J. F. Bergmann, 1905.

5. Stenger: Beitrag zur Kenntnis der nach Kopferletzungen affretenden Veranderungen im inneren Ohr, Arch. f. Ohrenh. **79**:43, 1909.

6. Amberg, Emil: Injuries to the Head, and Ear Disturbances, J. Michigan M. Soc. **13**:84, 1914.

7. Mygind, S. H.: Traumatic Vestibular Diseases, Acta oto-laryng. **1**:513, 1918-1919.

latter observation was somewhat higher. Mygind went on to observe that the postrotatory nystagmus as observed in 78 persons "seldom showed pathologic reactions." In the series herein reported, only 13 of the 35 persons on whom the rotation test was carried out demonstrated what would approximate a normal postrotatory nystagmus. At his hands, caloric examination in regard to both nystagmus and past pointing again failed to elicit as high a percentage of abnormal observations as we have encountered. He stated, "the pointing test after postrotatory and caloric experiment often showed only modified or an unaltered spontaneous (sic) deviation." In contrast, practically 100 per cent distortion of the postcaloric or postrotatory past pointing was found in our series. Induced vertigo or motion sensing, as a rule, was absent or shortened. Constitutional symptoms or responses from the vegetative nervous system, in the form of nausea, sweat or pallor, were also usually absent. Spontaneous vertigo accompanied by nystagmus could often be elicited by movements of the head. He also distinguished between the various types of equilibratory disturbances. He agreed with us in the observation that the posttraumatic vestibular symptoms are seldom accompanied by demonstrable involvement of the other cranial nerves. A further observation in accord is the frequency of delayed vestibular symptoms following injury, and likewise the decreasing irritability of the vestibular organ to artificial stimulation in subsequent examinations. The prognosis for recovery from vertigo is said to be good. This statement is contrary to our experience. He noted that this symptom is especially manifest when any work is undertaken which entails stooping or working at high altitudes. The statement is made that only one case of the series showed vestibular symptoms suggestive of cerebellar injury. We are inclined to believe, on the contrary, that a large percentage of postconcussional labyrinthine symptoms are due to injury to this part of the brain. This will be discussed presently.

Gottlieb[8] mentioned the lack of relation between the return of auditory function and the disappearance of vertigo. He pointed out abnormalities in past pointing in two cases in which at the same time were found horizontal canals responsive to the rotation test in the presence of markedly hypo-active responses from the vertical canals to douching. We have frequently noted this phenomenon.

Fletcher,[9] in a review of the subject in 1922, stated that the only way to determine the difference between psychogenic and organogenetic postconcussional vertigo is by means of neuro-otological tests. Hof-

8. Gottlieb, Mark J.: A Report of Two Cases of Head Injury, with Abnormal Oto-Neurological Findings, Laryngoscope **32**:783, 1922.

9. Fletcher, Harold A.: Determination of Disability as to Loss of Hearing, and the Importance of Vertigo: In Industrial Accident Cases, J. A. M. A. **79**:529 (Aug. 12) 1922.

mann [10] concluded that the phenomena noticed after concussion are due to injury to the end-organ itself rather than to central involvement. As will be seen later, we do not subscribe to this view. Brünner's [11] comprehensive work will be reviewed elsewhere in this paper.

Schuster [12] estimated that "forty per cent of all head injuries produce ear symptoms. These are impaired hearing, dizziness, tinnitus and nystagmus." His statement that the caloric tests in these cases "reveal normal irritability" is in direct contrast with our observation that in nearly all instances, at least in those in which concussion has been demonstrated, it has been possible to elicit abnormal postcaloric vestibular observations.

Osnato and Giliberti,[13] in a recent analysis of 100 cases of concussion, grouped dizziness and giddiness under one heading and found it present in fifty-seven of their cases. We place this observation at about 90 per cent.

Grove,[14] in a recent comprehensive survey of the subject, remarked that postconcussional vertigo, if of vestibular origin, is characterized by systematized dizziness accompanied by nystagmus brought on by movements of the head. He classed other types of equilibratory disturbance as of neurotic origin. His observations of abnormal responses after stimulation are considerably lower than our experience would warrant. He expressed the belief that most spontaneous phenomena of postconcussional vertigo are due to a decompensation or imbalance between the two labyrinths.

VERTIGO

Vertigo has been described as a disturbance in the relationship of one's self to external objects in space. We shall not attempt to adhere strictly to the letter of this definition. It is doubtful if most of the disturbances of the equilibratory apparatus, as described by people who have suffered from cerebral concussion, could be covered by this description. Most of them will not admit of experiencing any systematized motion sensing. They describe their sensations variously, as a "giddiness," "unsteady on feet," "tendency to fall in various directions without giddiness," "wavering of objects," "feeling as though head

10. Hofmann, L.: Lesion labyrinthique avec dissociation nystagmique consecutive à un traumatisme cranien, Ann. d. mal. de l'oreille du larynx **44**:1254, 1925.

11. Brünner, H.: Pathologie und Klinik der Erkrankungen des Inner-Ohres nach Stumpfenshadeltraumen, Monatschr. f. Ohrenh. **59**:697, 763 and 922, 1925.

12. Schuster, F. B.: Head Injuries with Ear Symptoms, Southwestern Med. **11**:116, 1927.

13. Osnato, M., and Giliberti, V.: Postconcussion Neurosis—Traumatic Encephalitis, Arch. Neurol. & Psychiat. **18**:181 (Aug.) 1927.

14. Grove, W. E.: Otologic Observations in Trauma of the Head, Arch. Otolaryng. **8**:249 (Sept.) 1928.

goes up and down," "dazed feeling," "sensation as if going up in an elevator," "objects receding," "mental confusion, especially in crowds or under any unusual stress, or on looking upward," or that their heads "sway like a swing." The majority of these sensations are not to be classed as true vertigos. Only four of the thirty-six patients described what could be considered as true motion sensing, i. e., of experiencing the sensation of objects revolving around them in a definite manner. Of interest is the fact that the vertigo induced at the time of the vestibular tests rarely corresponded in type to the vertigo of which the patient complained as a symptom.

Vertigo, or motion sensing, may be divided into two types:

1. True or systematized vertigo, in which external objects appear to revolve around the subject in a definite direction. This type of motion sensing is usually considered to originate in the end-organ and is often associated with spontaneous labyrinthine phenomena, such as nystagmus, past pointing and characteristic disturbances in station.

2. False or nonsystematized vertigo, which may be simply a feeling of uncertainty or confusion. The latter form of equilibratory disturbance may be caused by ocular disturbances or intracranial lesions involving the vestibular tracts, or it may be due to so-called neurasthenic vertigo. In other words, it does not originate in the end-organ, but may be the result of a derangement of the ocular, tactile or joint muscle sense or a combination of any of these.

According to Hubby,[15] the latter type of equilibratory disturbance is the result of interference with the intracranial vestibular pathways. Conversely, vertigo originating in the end-organ is usually associated with spontaneous labyrinthine phenomena, especially during the early state of the disturbance.

That these posttraumatic equilibratory disturbances are of prolonged duration is shown by the following facts: Twenty-seven patients showed little or no improvement in the subjective vertigo over periods of observation extending from nine days to twenty-four months. The vertigo was stated to become progressively worse in one instance. Seven patients claimed improvement. As before intimated, we have several times observed a progressive decrease in induced vestibular responses coincident with an improvement in the vertigo.

PATHOLOGIC CONSIDERATIONS

The pathologic processes that bring about posttraumatic vertigo naturally fall into two large divisions: (1) injuries to the inner ear, and (2) injuries to the eighth nerve, its intracranial pathways and nuclei. Brünner stated that one can have concussion of the brain without having concussion of the inner ear, but one cannot have concussion

15. Hubby, Lester Mead: The Modus Operandi of Vertigo, Arch. Otolaryng. 6:405 (Nov.) 1927.

of the inner ear without having concussion of the brain. Our experience indicates that in nearly all instances the vestibular symptoms following concussion are due to a combination of end-organ and central injury. Brünner produced concussion experimentally by striking animals on the head with varying degrees of force. He found postmortem evidence in the form of blood and fibrin in the cochlea and ductus cochlearis, serous exudate, edema of the organ of Corti and hemorrhage into the nerve. He also reported the presence of hemorrhage into the internal auditory meatus and exudate in the cochlea and vestibule in persons who had met death as a result of injury to the head. He summarized his data both from experimental and from clinical observations as follows:

> In experimental concussion of the brain, there are found, as a rule, degenerative changes in the nuclei of the cochlear and vestibular nerves—especially the latter. These degenerative changes last as long as the animal survives the trauma. The changes in the brain are partially due to the direct effect of the trauma on the brain tissue and partially to the circulatory disturbances brought about by the trauma within the skull. The prognosis in cases of concussion is made with the understanding that periodic attacks of vertigo persist for many years. It is possible to demonstrate concussion of the brain with symptoms in the ears by vestibular observations and symptoms. The observations in experimental concussion are manifest mostly in the blood vessels, in the perilymph and endolymph, in the aqueductus cochlearis, and in the formation of connective tissue and the occurrence of hemorrhage. If there is an impairment in auditory function following concussion, the prognosis is poor for the return of hearing.

Concussion of the inner ear, in conjunction with concussion of the brain, is probably brought about by the same mechanical means. Brünner assumed the establishment of a prestatic circulation with the formation of exudate in the structures of the inner ear. The latter has been termed otitis interna serosa, or otitis interna vasomotoris.

Injuries to the eighth nerve itself, in the form of hemorrhage or lacerations, are probably more common than injuries to other cranial nerves, with the probable exception of the seventh. An explanation would be offered by the fact that these two cranial nerves, shortly after leaving the brain stem, enter the internal auditory meatus. This anatomic consideration would lend plausibility to the theory that the eighth nerve may be pinched or injured at its point of entrance into the fallopian canal in cases of concussion of the brain. The assumption would be that the brain stem has been displaced by the impact of the injury. In addition to this, the fact that the neurons of the eighth nerve are peculiarly vulnerable to toxins, such as syphilis (Linthicum[16]), would also suggest that they might be particularly susceptible to trauma.

16. Linthicum, F. H.: Neuro-Otologic Studies in Central Nervous System Syphilis, Tr. Pacific Coast Oto-Ophthalmological Society, 1923.

In the present series the preponderance of evidence, as judged by neuro-otological studies, points either toward injury to the inner ear in conjunction with its intracranial pathways or to damage to these pathways alone. We have not found evidence pointing to isolated injury to the inner ear following concussion. As evidence of central involvement are the following observations: the frequent occurrence of a much diminished, or even absent, past pointing after stimulation, in the presence of hyperactive nystagmus; the induction of perverted nystagmus; the infrequent presence of spontaneous past pointing and nystagmus; in relatively few instances, the production of a postural nystagmus; postural vertigo, that is, attacks of dizziness with the head in certain positions, frequently noted without a simultaneously occurring nystagmus; the rare correspondence of spontaneous disturbances in equilibrium described by the patient to the vertigo produced by artificial stimulation of the labyrinth, and the observance that the severity of the symptoms complained of are usually out of all proportion to the degree of the spontaneous phenomena observed.

Another more or less consistent observation suggesting central involvement is the frequency of marked vestibular derangement in the presence of normal or but slightly impaired hearing. It is improbable that there would occur concussion of the inner ear alone sufficiently severe to alter the vestibular responses without at the same time interfering to some extent with auditory function.

Again, one of the most consistent observations throughout the series was the failure of the person being examined to past point properly after stimulation by either turning or douching. Of all of the vestibular responses to which we may attach any significance as diagnostic measures, past pointing is least understood. In a general way, absent past pointing, or crossed pointing after stimulation, is supposed to be suggestive of cerebellar involvement. It was interesting to note that in one instance a person examined shortly after an accident showed an entire absence of past pointing in the presence of hyperactive nystagmus. Reexamination a few months later showed the past pointing still to be absent and the nystagmus to have disappeared completely. There is some evidence to show that impulses for vertigo aroused by artificial stimulation are relayed through the cerebellum in their transmission from the ear. The universal occurrence and persistence of vertigo, together with this more or less constant perversion or absence of past pointing in cases of concussion, suggests that in addition to damage to other central structures, we are dealing as well with cerebellar injury or irritation. Greenacre [17] reported fifteen cases of traumatic pontile

17. Greenacre, Phyllis: Bull. Johns Hopkins Hosp. **28**:86, 1917.

hemorrhage in which points of punctate bleeding were found to be grouped around the periphery of the pons. Such injuries to the pons and cerebellum suggest an explanation for the high percentage of abnormal past pointing.

AUDITORY PHENOMENA

Functional hearing tests were carried out in thirty-two instances. The auditory phase will be considered only in its relation to the vestibular picture. There were only six persons who failed to show some impairment of the receptor or inner ear type, manifest by the usual shortening of bone conduction and loss of acuity for high tones. Of this group, the impairment was unilateral in eleven instances and bilateral in the remaining fifteen. Of the six cases in which there was no manifest auditory impairment, only one failed to exhibit well defined vestibular abnormalities.

The auditory impairment coincided with the vestibular abnormalities in twenty examinations and disagreed in twelve. By this is meant that the auditory damage was not always homolateral with the more marked vestibular involvement. In the cases that were rechecked, the hearing was found to have improved in three, retrogressed in two and remained unchanged in two. In no instance, however, was there any corresponding change in the vestibular responses, except for the worse, and on only one occasion was the auditory acuity markedly reduced without a corresponding distortion of the vestibular reactions.

POSTCONCUSSIONAL CEREBELLOPONTILE ANGLE SYNDROME

Probably the most frequent intracranial condition to be diagnosed by means of vestibulo-auditory tests is the so-called cerebellopontile angle syndrome. The lesion either may be an acoustic tumor arising from the eighth nerve itself, or may be brought about by invasion of the angle by new growths having their origin in the cerebellum or pons. Accumulations of fluids or cysts may give a similar picture. The syndrome, in brief, is as follows: on the affected side there are usually tinnitus and impairment of hearing of the receptor or inner ear type; all of the canals fail to respond to douching. On the opposite side, there is no tinnitus or impairment of hearing; the horizontal canal alone responds to douching. We have encountered six instances of concussion which simulated in whole, or in part, the typical cerebellopontile angle syndrome. From a neuro-otological standpoint, the chief point of difference between the two conditions was the fact that in the post-traumatic cases there was no constant impairment of auditory function. In pontile angle lesions, especially in tumors of the acoustic nerve, a history of early unilateral auditory impairment can usually be elicited

which, on functional tests, proves to be of the receptor type. The relation between the auditory and vestibular observations in the six cases of simulated pontile angle tumor is as follows:

Auditory Symptoms	Vestibular Symptoms
Bilateral impairment; receptor type; equal on the two sides; tinnitus present, not lateralized.	All the canals on the left side and the right vertical canals fail to respond. The right horizontal canal is active.
Bilateral impairment; receptor type; more marked on the right side; tinnitus absent.	All canals on the left side and the right vertical canals fail to respond. The right horizontal canal is active.
Bilateral impairment; receptor type; equal on the two sides; tinnitus present, not lateralized.	All the canals on the left side and the right vertical canals fail to respond. The right horizontal canal is active.
Bilateral impairment; conductive type; more marked on the right side; tinnitus lateralized to the right side.	All the canals on the right side and the vertical canals on the left side fail to respond. The left horizontal canal is active.
Bilateral impairment; receptor type; complete on right side; tinnitus present; not lateralized.	All canals on the right side and the vertical canals on the left side fail to respond. The left horizontal canal is active.
Right-sided impairment; receptor type; tinnitus (?).	All canals on the right side and the vertical canals on the left side fail to respond. The left horizontal canal is active.

It will be seen that in the postconcussional angle syndrome, cochlear impairment is by no means so constant as in the other types enumerated. Consequently, the determination of the degree of auditory damage, together with a careful history, makes it possible to differentiate between the two conditions. What pathologic changes bring about this picture in posttraumatic cases? One could assume a collection of fluid, organized clot or some similar lesion in the region of the pontile angle on the affected side. Why such a lesion, the nature of which is not clear, should affect the vertical canals on the opposite side, we can no more surmise than we can in cases of tumor of the acoustic nerve. These observations following injuries to the head emphasize the importance of an accurate history and complete auditory examination before making a diagnosis of a cerebellopontile angle tumor purely from a neuro-otological study.

A similar case was reported by Zachs.[18]

HISTORIES

Independent neurologic and neuro-otological histories and examinations were obtained in practically all instances. In these inquiries,

18. Zachs, Myron A.: Interesting Cochlear and Vestibular Findings Following Accident, Laryngoscope **38**:288, 1928.

special stress was laid on the following points: Whether or not there had been any previous auditory or vestibular disease; whether or not the patient was unconscious following the accident; on what part of the head the injury was sustained; whether there was bleeding from one or from both external auditory canals; the interim after the injury when vestibulo-auditory symptoms appeared; whether the symptoms were brought on by postural changes; whether or not the vertigo was systematized; whether the symptoms were progressive or retrogressive; the presence or absence of disturbance in gait or station; visual disturbances, and whether or not there was an accompanying tinnitus and nausea at the time of the attacks. Too much stress cannot be laid on the accuracy of the history, as it plays no small part in the final determination of the extent of injury and the consequent prognosis.

SPONTANEOUS PHENOMENA

As spontaneous phenomena we list nystagmus, past pointing and disturbances in station, which occur independently of stimulation in the form of turning or douching. The observance of spontaneous phenomena after concussion is remarkably small. We encountered spontaneous nystagmus in ten patients, horizontal nystagmus in seven and rotatory nystagmus in three. It must be borne in mind that spontaneous nystagmus may be present at one examination and absent at the next. Its presence, or absence, depends largely on how soon after injury the inspection is made. Children and young adults seem to show spontaneous nystagmus following injury more frequently than older persons. Vertical nystagmus has not been observed.

The majority of patients who exhibited a spontaneous past pointing were found to have hyperactive responses on rotation. This did not hold for the caloric test. Spontaneous past pointing occurred much more frequently than spontaneous nystagmus, being observed seventeen times. It was usually in the direction of the more affected ear. Only six of the persons exhibiting spontaneous nystagmus were found to have spontaneous past pointing. Spontaneous falling was observed in fourteen instances. Only twice was this disturbance in station of the so-called end-organ type observed. By this is meant, given, for example, a lesion in the right inner ear, the person tested is supposed to fall toward this ear regardless of the position in which the head is placed; i. e., with the face turned toward the right shoulder, he will fall backward; with the face turned toward the left shoulder, he will fall forward. In only two instances of this so-called spontaneous falling was there a coincident spontaneous nystagmus. There were six instances in which the falling was always in the same direction, regardless of the position of the head. This is interpreted as implying a central rather than a

peripheral involvement. The foregoing differentiation of the two types of falling is merely suggestive, and too much significance should not be placed on its presence or absence.

In the cases rechecked, spontaneous nystagmus and past pointing were not found to be constant; that is, they would either disappear or reappear intermittently. Disturbances in station were found to be apparently unaffected by the passage of time, and they remained the same throughout all subsequent examinations. Postural nystagmus, by which is meant the induction of nystagmus by sudden changes in the position of the head, was elicited in only one instance. In the three patients in whom there was a falling reaction of the so-called end-organ type, the spontaneous past pointing was to the same side. On the contrary, in the instances in which the falling was of the so-called central type, the past pointing was either absent or apparently bore no relation to the direction in which the falling took place.

INDUCED NYSTAGMUS

Postrotatory Nystagmus.—In general, it may be said that the nystagmus after turning approached much nearer to normal, in direction, quality and amplitude, than any of the other responses. In these tests we employed the routine procedure of turning the patient ten times in twenty seconds with the head inclined 30 degrees forward. We took as normal an after-nystagmus of from twenty to twenty-four seconds' duration. A longer or shorter duration was considered as hypo-active or hyperactive, respectively.

Of the thirty-five persons rotated, hyperactive nystagmus was encountered seven times; normal nystagmus was present in thirteen, and hypo-active responses were obtained in fifteen instances. On only one occasion did we encounter an entire absence of postrotatory nystagmus. Discrepancies in the duration of the nystagmus from the two sides in the same person varied from five to fourteen seconds and occurred twelve times. One would expect the most marked differences in responses from the two sides to appear when the examination was carried out shortly after the injury, before so-called vestibular compensation had commenced. This was found to be the case. In three of our cases in which a marked discrepancy in postrotatory nystagmus was observed at the time of the initial examination, this compensation was found to have taken place when the patients were subsequently tested. For example, patient C-3 (table 2, case 1) was rotated six months after injury. Turning to the right elicited a horizontal nystagmus of twenty-five seconds' duration; turning to the left, a nystagmus of sixteen and one-half seconds' duration. Three months later the nystagmus was of fifteen seconds' duration on each side.

Experimentally, it has been shown that if the labyrinth on one side is destroyed and the rotation test is carried out soon afterward, there will be an approximately normal response from the uninjured side and a shortened or absent response from the destroyed side. Conversely, if in from nine months to a year the subject is again tested, it will be found that his vestibular responses will be approximately equal from the two sides, with the difference that the duration of the nystagmus from each side will be approximately one-half the normal.

In marked contrast to the generally active functioning of the horizontal canals to the turning test was the frequent failure of the same canals to produce nystagmus after douching. The caloric test has the advantage of determining the function of each inner ear individually, as distinguished from the rotatory procedure, in which both labyrinths are stimulated at the same time. After douching it was frequently noted that a normal nystagmus was obtained from one side in the presence of an absent, decreased or perverted nystagmus from the opposite side. By perversion in this instance is meant either a rotatory or a vertical nystagmus where a horizontal nystagmus should obtain, or conversely. In five of the seven cases which were followed by vestibular reexaminations, the postrotatory nystagmus was found to decrease in duration on subsequent examinations. In these there was apparently a loss of responsiveness to stimulation as time elapsed after injury. In the cases in which it was shown by the caloric test that one horizontal canal was functioning to the exclusion of that on the opposite side, especially when the examination was carried out soon after the injury, one would expect that postrotatory stimulation would bring out a similar difference between the two sides. This was not found to be the case. In two instances in which the vestibular tests were carried out four months after injury, the postrotatory nystagmus was apparently equal and normal on the two sides, while douching revealed a definite perversion of response from one horizontal canal, with the opposite side showing a normal reaction. In most instances, however, the responses from the horizontal canals, both for douching and for turning, were consistent. The two instances cited, however, bring up the much debated question as to whether or not one stimulates the same part of the end-organ by turning as by douching, or, at least, whether one stimulates it in the same manner.

Caloric Nystagmus.—The technic for the caloric test was carried out by mass irrigation; i. e., with the patient in the erect position and the head inclined 30 degrees forward, the ear was douched with water at 68 F. until a nystagmus became manifest. The period elapsing between the beginning of the irrigation and the appearance of the nystagmus was noted. The irrigation was then continued for three minutes, if the condition of the patient permitted. The head was then tilted back, and

the nystagmus again noted. Past pointing was carried out with the head in both positions, and the presence or absence of vertigo and constitutional symptoms observed.

As a rule, it may be stated that the vertical canals were either nonresponsive or hyporesponsive to the caloric test. In thirteen persons there was no postcaloric nystagmus from the vertical canals from either side. In ten it was present on one side and absent on the other. When this condition obtained, the nystagmus on the responsive side generally, if not always, was delayed in making its appearance. It was hyperactive in five instances on both sides and in one case on a single side. In only four tests could the responses from the vertical canals, as regards nystagmus, be considered within normal limits. In nine of the examinations the horizontal canal was found to give normal responses on the same side on which it was not possible to elicit responses from the vertical canals.

PAST POINTING

Abnormal past pointing was the most consistent of all observations; only twice in the series could the past pointing after turning be considered within normal limits. The most frequent aberration in this test was the repeated occurrence of crossed past pointing. By this is meant a deviation, after turning, of one or both arms in a direction opposite to the one to be expected. It was frequently absent in one or both arms, or absent in one arm and crossed in the other, or again entirely absent in both arms. Analysis of past pointing, as related to nystagmus after rotation or douching, is practically impossible.

When testing for past pointing it must be remembered that the subjective element plays a much larger rôle than it does in the nystagmic responses, or in the so-called constitutional symptoms. Nevertheless, even when this factor is discounted, one cannot but be impressed by the remarkable and consistent perversion of past pointing as a postconcussional phenomenon. Hardly less constant are the absent or subnormal constitutional symptoms. A normal person whose ears are douched with cold water will, within approximately a minute, become actively ill, or at least show signs of distress arising through stimulation of the vegetative nervous system. The failure of nausea, sweat and pallor to occur, even after prolonged stimulation and often in the presence of an active nystagmus, again suggests the possibility of intracranial derangement of the vestibular mechanism in concussion.

RECHECKED CASES

It was possible to follow with reexamination six cases of concussion over periods varying from three to twenty-four months. These cases deserve discussion in some detail. They are summarized in table 2.

The first patient (C-3) was seen six months after injury. The initial examination revealed a complete deafness of the inner ear on the left side with rather marked auditory impairment on the right side. He showed no spontaneous nystagmus, but exhibited a tendency to fall to the left. He past pointed spontaneously to the right with the right arm and to the left with the left arm. After turning, there was an active horizontal nystagmus from both sides, which was shorter after turning to the left. After douching, the right vertical canals responded normally, while the left ones produced a nystagmus only after two minutes. When the head was put back, the nystagmus from the left horizontal canal was found to be perverted. Past pointing was absent in the right arm after douching the left ear. He was tested again three months later. Auditory function on the left was still absent; there was some evidence of return of hearing on the right side. The spontaneous observations remained unchanged. Nystagmus after turning to the right had dropped to fifteen seconds, but was found to be the same after turning to the left. The nystagmus after douching of the left ear had disappeared from both the horizontal and the vertical canals. The other observations remained the same. He was checked a third time a year after his accident. The auditory observations were the same. There was no change in the spontaneous phenomena. The nystagmus and past pointing after turning and douching were the same as on the last examination. Five months later he was again tested. There was a slightly lessened irritability of the right labyrinth; subjectively, there was no improvement. His next examination was four months later. There was no change worthy of comment in the vestibular picture. He was still troubled with vertigo to the extent that he could not work. On the final examination, his condition had apparently become stationary.

This patient had six vestibulo-auditory examinations carried out over a period of nearly two years. The history was that of an injury over the left mastoid process, which rendered him unconscious. He complained of deafness immediately following the accident and dizziness; the latter was precipitated chiefly by sudden movements of the head to the left. He had a "roaring" tinnitus which was aggravated during his attacks of vertigo. Over the period of examinations, there was no improvement in his subjective symptoms as first described. There was no demonstrable change in his hearing. The results of vestibular tests remained practically unchanged throughout the period of observation except to show a slight decrease in the ability of the right labyrinth to respond to the tests and a negligible return of function in the left labyrinth. At the end of two years his vertigo was such as to incapacitate him for work.

The second patient (C-50) was examined three times over a period of five months. He was first seen about nine months after his accident. He was unconscious for ten minutes. At the end of two weeks he attempted to work, but was unable to continue on account of dizziness and nausea, which were aggravated by quick movements of the head to one side. At the end of another week he again attempted to work. He then noticed that his gait was uncertain and that he tended to deviate to the right. At the end of five months, during which time he remained idle, avoiding any exertion, his subjective symptoms had improved. Although there was an occasional slight uncertainty of gait, he no longer deviated toward the right. Quick movements still caused blurring of vision, which was less marked than at first. Objectively, there was a decrease in the vestibular responses to stimulation. The picture changed from a state of hyperactivity of the left labyrinth after douching to one of hypo-irritability. The spontaneous nystagmus seen on the first examination had disappeared. The auditory picture remained the same.

We here note an improvement in the subjective symptoms coincident with a decrease in vestibular function.

A third patient (C-59) was first examined one month after his accident and again five months later. His auditory impairment was of mixed conductive and receptor type. It had become more marked at the time of his second examination. The spontaneous past pointing and falling, which were present at the initial examination, were absent subsequently. The nystagmus after turning showed a decrease in response from the left horizontal canal at the second examination, while the caloric nystagmus dropped out entirely after having shown fairly good responses at the first test. In like manner the past pointing subsequently disappeared. The whole picture showed a decrease in labyrinthine responsiveness and an improvement in the subjective symptoms of vertigo and uncertainty in gait.

The fifth patient (C-67) was examined at periods of two months and five months subsequent to his injury. There were no spontaneous observations and slight auditory impairment. The nystagmus after douching of the right ear was normal at the first examination and had disappeared at the second examination. The left ear showed marked hypo-irritability on a second examination. Past pointing was absent throughout after the caloric tests.

The sixth patient (C-2) was first seen four months after his injury, again in a year and a third time nine months later. There was an apparent complete nerve deafness on the right side and moderate impairment on the left. On the final test, some auditory improvement was noted on both sides. At all times vestibular responses were absent on the right side. The vestibular picture remained the same while he was under observation; subjectively, there was considerable improvement.

There were two facts to be noted in the six cases: 1. There was a gradual loss of labyrinthine function as time elapsed after the accident. 2. Coincident with this deterioration, a readjustment of the subjective symptoms of equilibratory disturbances took place up to a certain point.

In analyzing this series, we hoped to establish some definite neuro-otological picture that would indicate whether postconcussional labyrinthine symptoms could be demonstrated by objective neuro-otological observations and whether these observations could be grouped in such a manner as to enable one to offer a prognosis for recovery. We have found that definite vestibular abnormalities can be demonstrated in most postconcussional cases. We have, however, been unable to group these abnormalities into a definite symptom complex, except that it would seem that the vertigo resulting from injury to the end-organ itself is much more liable to readjustment than are the various equilibratory disturbances due to damage to the intracranial pathways. Consequently, if one can determine at the time of examination whether peripheral or central involvement is more in evidence, one is justified in attempting a prognosis. In other words, if central damage predominates, the prognosis is poor; if the end-organ is at fault, the outlook is better.

RÉSUMÉ OF CASE REPORTS

CASE 1 (County).—*Severe concussion.*—A man, aged 59, a laborer, was admitted to the Los Angeles General Hospital on Oct. 12, 1926, after he had been struck by an automobile while working on the road. He remained unconscious for two days. There was no bleeding from the ears. Roentgenograms of the skull showed no fracture. The Wassermann reaction of the blood was negative. Neurologic examination gave essentially negative results, save for hyperactive knee reflexes. No pathologic reflexes were elicited. There were no pupillary or fundus changes.

On Feb. 3, 1927, the auditory test (audiometer) showed: receptive type of lesion on the left side; the vestibular test: spontaneous rotatory nystagmus to the left; delayed responses from the right vertical canals; oblique nystagmus from the right horizontal canal.

CASE 2 (C-16).—*Linear fracture of the left parietal bone; concussion.*—A man, aged 30, a rotary helper, was referred by Dr. W. T. Cade, Jr., on Feb. 4, 1926. He was injured on Nov. 17, 1925, when he was struck on the head by a falling timber. He was rendered unconscious for half an hour, and bled profusely from the left ear. He returned to work two months later, but complained of generalized headache and dizziness on stooping. Three months after the injury, neurologic examination gave essentially negative results, except for a dilated left pupil and unsteadiness of the eyes on looking to the right. There had been almost total deafness in the right ear since childhood; the hearing were impaired on the left, and Weber's test was referred to the left. Tests with the tuning fork showed the bone conduction better than the air conduction on the right side, and the air conduction better than the bone conduction on the left. Roentgenograms of the skull showed a linear fracture in the left posterior parietal region extending to the external auditory canal. The Wassermann reaction of the blood was negative.

On Feb. 17, 1926, the auditory test showed: receptive type of deafness on the right; the vestibular test: absent responses from all canals on the right; horizontal canal on the left functioning; a picture of angle tumor.

CASE 3 (C-35).—*Mild concussion.*—A man, aged 33, an interior decorator, was referred by Dr. J. Rollin French. On April 15, 1926, while working on a scaffold, he bumped the top of his head against a beam of the ceiling. He was not rendered unconscious, and did not fall. He worked the rest of that day and for three days following; he then complained of headaches, poor memory, feeling of flightiness, insomnia and inability to work. Roentgenograms of the skull were negative. Neurologic examination, made by Dr. Paul E. Bowers, gave essentially negative results, except that the knee reflex and achilles tendon reflex of the right side were quicker than those on the left.

On July 28, the auditory test showed: conductive type of lesion, bilateral; the vestibular test: left horizontal canal, the only one to respond to douching; abnormalities in past pointing; neurologic picture of angle lesion without typical auditory observations.

CASE 4 (C-48).—*Concussion; neurosis.*—A man, aged 50, a laborer, was referred by Dr. John D. Gillis on June 7, 1926. He was injured on Feb. 2, 1926, when he fell from a truck to the cement floor, striking the back of his head. He was not rendered unconscious, and there was no bleeding from the ears. He remained in bed for two days. Subsequently, he began to have occipital headaches and dizziness, which persisted and became worse during the next eight months. He complained of pain in the region of the right great occipital nerve, and a mild neurosis developed. Roentgenograms failed to reveal a fracture of the skull. The Wassermann reaction of the blood was negative. Neurologic examination gave negative results, save for evidences of a neurosis. Eight months after the injury he was still unable to work.

On December 22, the auditory test showed: bilateral lesion of the mixed receptive and conductive type; the vestibular test: absent responses from the vertical canals on both sides; some function in the right horizontal canal; resembled vestibular observations in cases of angle tumors.

CASE 5 (C-38).—*Severe concussion.*—A man, aged 28, a street decorator, was referred by Dr. J. Rollin French. On July 25, 1926, he fell about 25 feet from a ladder to the street. He had no recollection of falling, and remained unconscious for a week. Six weeks later he complained of dizziness, sinking spells, "sensation around my forehead which robs me of my energy," loss of memory, inability to study and headaches. The neurologic examination made by Dr. C. G. Johnson, was said to be negative, but the patient showed a peculiar mental make-up simulating dementia praecox. He swayed in Romberg's position. There was no bleeding from his ears. Roentgenograms of the skull showed no fracture.

On September 8, the auditory test showed: very slight receptive type of lesion, bilateral; the vestibular test: responses from the horizontal canals subnormal after turning; no response from the vertical or horizontal canals after douching.

CASE 6 (C-0).—*Linear fracture of the right parietal region; concussion and contusion of the brain.*—A man, aged 24, an oil worker, was referred by his employer on June 15, 1927. Two months previously he was thrown from his automobile, and was rendered unconscious; he remained so for three days. There was marked bleeding from the right ear, and a right peripheral facial paralysis immediately followed. Three months later he complained of headache, dizziness on exertion, ringing in the right ear and weakness of the right side of the face. Neurologic examination gave negative results, except for dilatation of the right

pupil and a complete right peripheral facial paralysis. The sense of taste was lost over the right anterior two thirds of the tongue. He swayed to the right in Romberg's position. Roentgenograms of the skull showed a linear fracture in the right parietal region running toward the external auditory meatus.

On July 14, the auditory test showed: no function in the right ear; receptive type of lesion in the left ear; the vestibular test: marked impairment; no responses from the right side and only the horizontal canal functioning on the left side; typical picture of an angle tumor.

CASE 7 (C-60).—*Concussion; neurosis.*—A man, aged 54, a carpenter, was referred by Dr. W. T. Cade, Jr., on Oct. 14, 1926. On Sept. 7, 1926, he was struck on the right side of the head by a piece of timber which fell 10 feet. He remembered being hit, but soon became unconscious, remaining so for four hours. A hematoma developed, and he was told that he had a depressed fracture of the skull and that operation would be necessary. Five weeks later, he complained of headaches, dizziness and weakness. He was nervous and shaky. Neurologic examination revealed some overfilling of the retinal veins, an increase of the right knee reflex and active vasomotor disturbance. Marked neurosis was present. Roentgenograms of the skull were negative for fracture. The Wassermann reaction of the blood was negative. His neurotic symptoms remained unchanged for about a year.

On July 7, 1927, the auditory test showed: receptive type of lesion, bilateral, and more marked on the left side; the vestibular test: absent past pointing in the right arm after douching of the left ear; subnormal constitutional responses.

CASE 8 (C-66).—*Concussion.*—A man, aged 35, a truck driver, was referred by Dr. John N. Osburn and Dr. G. F. Boehme, Jr. He was injured on Dec. 4, 1926, in a collision, when he was thrown from his truck. He was unconscious for several hours. Two weeks later his principal complaint was bilateral deafness. The results of the neurologic examination, by Dr. G. F. Boehme, Jr., were reported as negative except for impaired hearing. Roentgenograms of the skull showed no fracture. The question of hysterical deafness or malingering arose.

On Aug. 11, 1927, the auditory test showed: conductive type of lesion on both sides, more marked on the left; the vestibular test: total loss of responses after turning and douching.

CASE 9 (C-65).—*Mild concussion.*—A man, aged 20, a carpenter, was referred by Dr. J. Rollin French. He was injured on May 23, 1927, when he was struck by a falling plank and knocked backward, striking his head against a brace. He was not rendered unconscious. Ten days later, he stated that he could not hear with his right ear. Neurologic examination, made by Dr. C. G. Johnson on June 27, gave essentially negative results, except for impaired hearing on the right. Roentgenograms of the skull showed no fracture.

On July 8, the auditory test showed: bilateral, mixed conductive and receptive type of lesion, more marked on the right side; the vestibular test: the vertical canals did not respond on either side; abnormalities in past pointing.

CASE 10 (C-64½).—*Concussion.*—A man, aged 54, a carpenter, was referred by Dr. H. E. Southworth on April 23, 1926. He was injured on Dec. 19, 1925, when he was struck on the head by a falling piece of steel and knocked 20 feet from a ladder. He remained unconscious for three hours. Four months later he complained of headache, dizziness and light-headedness. Following a dizzy spell, a severe frontal headache developed. The neurologic examination gave negative results. Roentgenograms of the skull revealed no fracture. His dizziness appeared to be out of proportion with the severity of his injury. He was observed for

more than two years, during which time he did not work, and he stated that the dizziness did not improve.

On Aug. 29, 1927, the auditory test showed: receptive type of lesion on both sides, more marked on the left; the vestibular test: vertical canals nonresponsive to douching; abnormalities in past pointing after douching of the right ear.

CASE 11 (C-63).—*Depressed fracture of the skull; concussion and contusion of the brain.*—A man, aged 28, an attendant at an oil station, was referred by Dr. W. T. Cade, Jr., on July 13, 1925. He had sustained a compound, comminuted, depressed fracture of the skull in the left occipital region on May 3, 1925, when he was struck by an automobile and rendered unconscious. The depressed fragments of bone were removed as an emergency measure. There was bleeding from the left ear. He complained of headache, dizziness and impairment of hearing in the left ear. Two months after the injury neurologic examination showed an increase of all deep reflexes on the right side, as compared with those on the left, but otherwise gave essentially negative results, except for rather marked neurotic symptoms. He was observed for a period of three years, during most of which he was able to work, but continued to complain of headache, nervousness and dizziness.

On July 21, 1927, the auditory test showed: acuity practically within normal limits; the vestibular test: hyperactive vestibular responses from all canals; abnormalities in past pointing.

CASE 12 (C-47).—*Concussion.*—A man, aged 51, a miner, was referred by Dr. J. Rollin French. On Aug. 30, 1926, he was struck in the midfrontal region by a falling rock. He was dazed, but not entirely unconscious. There was no bleeding from the ears. He continued to work for about two weeks, when purulent otitis media developed on the right side, which continued for approximately four months. One month after the injury he complained of headaches, dizziness and insomnia. Neurologic examinations made by Dr. C. G. Johnson and Dr. Paul E. Bowers were reported as negative. Roentgenograms of the skull failed to reveal a fracture.

On November 24, the auditory test showed: conductive type of lesion on the right side; the vestibular test: delayed responses from the right vertical canals; oblique nystagmus from the right horizontal canal; no response from the left vertical canals; absent past pointing from the right arm; crossed past pointing with the left arm after douching of the left ear.

CASE 13 (C-44).—*Mild concussion.*—A man, aged 47, a laborer, was referred by Dr. W. T. Cade, Jr., on Oct. 27, 1926. He was injured on Oct. 18, 1926, when he slipped on a cement floor, striking the back of his head. He remained unconscious for a few minutes. His principal complaints at the time of examination were occipital headache and marked dizziness. Neurologic examination showed the pupils to be irregular, the left being slightly larger than the right; they reacted well. Hearing was impaired on the right. There was a suggestive Babinski sign on the left; otherwise, the reflexes were normal. Roentgenograms of the skull were negative for fracture. The Wassermann reaction of the blood was negative.

On October 22, the auditory test showed: slight impairment on the left, conductive type of lesion; the vestibular test: practically normal, except for absent past pointing in the right arm. This was the nearest approach to a normal reaction encountered in instances of concussion.

CASE 14 (C-43).—*Mild concussion.*—A man, a laborer, was referred by Dr. W. T. Cade, Jr., on Oct. 20, 1926. He was injured on Aug. 13, 1926, in a cave-in,

when a large lump of clay struck him on the left side of the face and neck. He was stunned for a short time. For several days he complained of dizziness, which became worse when he arose from a recumbent position. Two months after the injury, the dizziness was not prominent. Several days after the injury, he complained of deafness and a buzzing sound in the left ear; he disclaimed any previous ear trouble. Neurologic examination was not available.

On October 20, the auditory test showed: mixed conductive and receptive type of lesion on the left side; the vestibular test: absent responses; absent nystagmus from the right vertical canals; tendency to conjugate deviation from both horizontal canals; subnormal constitutional symptoms and abnormalities in past pointing after douching of the right ear.

Case 15 (C-42).—*Concussion.*—A man, aged 25, a pipe fitter, was referred by Dr. C. E. Gage. On Jan. 9, 1926, the patient was struck behind the left ear with a sledge hammer. He was rendered unconscious for about an hour. Following this he complained of headache, dizziness and deafness in the left ear. Neurologic examination showed an increase in the knee reflexes and a positive Babinski sign on the left, but gave otherwise negative results. There was impairment of hearing on the left side. Roentgenograms revealed no fracture of the skull.

On October 15, the auditory test showed: complete deafness on the left side; the vestibular test: abnormal and perverted past pointing.

Case 16 (C-30).—*Concussion.*—A man, aged 31, a laborer, was referred by Dr. W. T. Cade, Jr., on June 10, 1926. On May 7, 1926, he was struck on the back of the head by a falling bucket, and was rendered unconscious for four hours. There was no bleeding from the ears. He had violent headaches for the first few days, and six weeks later stated that "it felt as if the top of my head were coming off." He also complained of dizziness, which was severe after exertion. The head was tender over the great occipital protuberance. The neurologic examination gave negative results, save for sluggish pupils, a positive Romberg sign and unsteadiness and reeling in his gait. Roentgenograms of the skull failed to reveal a fracture.

On July 25, the auditory test showed: no impairment; the vestibular test: decreased responses from the right vertical canals and no response from the left vertical canals; abnormalities in past pointing.

Case 17 (C-21).—*Depressed fracture of the skull and left parietal bone; concussion and contusion of the brain.*—A man, aged 40, a clerk, was referred by Dr. H. E. Southworth on Feb. 20, 1925. He was injured on July 8, 1924, when he was struck on the left side of the head by a steel bar, being immediately rendered unconscious and remaining so for several hours. He sustained a compound, comminuted, depressed fracture of the skull in the left parietal region, and was operated on the same day. He showed complete aphasia and marked right hemiplegia. Both the hemiplegia and the aphasia gradually improved. Examination six months later showed some difficulty in speech and residual weakness of the right hand and the right side of the face. He complained of headache and dizziness. There were obtunding of smell on the left side and hypesthesia of the second division of the left trigeminal nerve. The fundi showed considerable new tissue in the optic cups and overfilling of the retinal veins. There was unsteadiness of the eyes on looking to the right and the left. The deep reflexes on the right side were increased as compared with those on the left. Roentgenograms of the skull showed a defect in the left parietal region.

On April 14, 1926, the auditory test showed: hearing tests not done; the vestibular test: right vertical canals failed to react to douching; no constitutional

symptoms, past pointing or vertigo in the presence of active nystagmus from the horizontal canals.

CASE 18 (C-20).—*Psychoneurosis; concussion* (?)—A man, aged 42, a laborer, was referred by Dr. C. E. Early on April 14, 1926. On May 25, 1925, he slipped on a concrete floor, striking the back of his head. He was not rendered unconscious and worked for the next four days. He then stopped because of headache, dizziness and impairment of hearing. Deafness was said to be greater on the right side than on the left. He claimed complete disability for a year following the accident. Four years prior to this he had injured his back, which had resulted in two and a half years of complete disability, the case going through Industrial Accident Commission channels. Neurologic examination showed a double internal strabismus with unsteadiness of the eyes on looking in various directions. The left pupil was larger than the right. He showed typical stocking anesthesia of both lower extremities of the hysterical type. Roentgenograms of the skull were negative for fracture. The Wassermann reaction of the spinal fluid and blood were negative.

On April 24, 1926, the auditory test showed: slight bilateral receptive type of lesion; the vestibular test: responses hyperactive after douching; abnormalities in past pointing.

CASE 19 (C-13).—*Concussion.*—A man, aged 40, a miner, was referred by Dr. Lawrence Chaffin. He was injured on Oct. 16, 1925, when a small steam shovel fell, pinning him between it and the wall of the mine. He was unconscious for a few minutes, but later walked out of the mine. On reaching the surface, he complained of blindness and ringing in his ears. The blindness cleared up in four days. On Feb. 2, 1926, he stated that he was totally deaf in the left ear. He had occasional attacks of dizziness. Neurologic examination, made by Dr. Chaffin on this date, gave negative results, except for deafness of the left ear. Roentgenograms of the skull showed no fracture.

On February 3, the auditory test showed: no function in the right ear; the vestibular test: the right vertical canals failed to respond to douching; absent past pointing in the presence of an active nystagmus.

CASE 20 (C-11).—*Separation of the coronal suture; concussion; syphilis.*—A man, aged 42, a laborer, was referred by Dr. H. E. Southworth on June 26, 1925. On Oct. 23, 1923, he was struck on the top of the head by a steam shovel, being rendered unconscious for about six hours. His principal complaints at the time of examination, three years later, were unsteadiness in gait, weakness of the right hand, dizziness, deafness of the right ear and tinnitus. Neurologic examination showed a spontaneous horizontal nystagmus on looking both to the right and to the left, moderate weakness of the right arm, marked ataxia of both upper and lower extremities, greater on the right, a strongly positive Romberg sign and a staggering gait. His deep reflexes were all sluggish, but equal. Roentgenograms of the skull showed slight separation of the coronal suture. The Wassermann reaction of the blood was strongly positive. Intensive antisyphilitic treatments had not helped him.

On Dec. 23, 1925, the auditory test showed: apparent total loss of the auditory function on the right side; the vestibular test: spontaneous nystagmus to the left: vertical canals nonresponsive on both sides to douching.

CASE 21 (C-9).—*Concussion* (?); *neurosis.*—A man, aged 50, a carpenter, was referred by Dr. W. T. Cade, Jr., on June 30, 1925. On March 30, 1925, he fell backward from a low scaffold, striking his head on a cement floor. He was stunned, but went back to work a few minutes later and finished the day. The

following day, he complained of dizziness. Three months later, he still had headache and dizziness. Neurologic examination gave negative results. The patient had obviously developed a neurosis. Roentgenograms of the skull showed no injury. The Wassermann reaction of the blood was negative.

On November 18, the auditory test showed: no abnormalities; the vestibular test: spontaneous nystagmus to the right; responses hyperactive; abnormalities in past pointing.

Case 22 (C-6).—*Concussion and contusion of the brain.*—A man, aged 60, a laborer, was referred by Dr. W. T. Cade, Jr., on Oct. 3, 1925. On Aug. 8, 1925, he fell through a causeway, being immediately rendered unconscious; he remained so for two weeks. Two months later, he complained of dizziness and bilateral deafness. He had no headache. Neurologic examination made on October 3, gave essentially negative results, save for impaired hearing on each side. Roentgenograms of the skull failed to reveal a fracture. The Wassermann reaction of the blood was negative. The patient was rather neurotic.

On October 5, the auditory test showed: no abnormalities; the vestibular test: no responses from the right side after douching; the left horizontal canal was functioning; a picture of angle tumor.

Case 23 (C-51).—*Linear fracture of the right parietal bone; concussion and contusion of the brain.*—A man, aged 40, a foreman on a ranch, was referred by Dr. W. T. Cade, Jr., on June 3, 1926. He was injured on Feb. 14, 1926, when his automobile went over an embankment. He remained unconscious for several hours, and was delirious for a month following. There was bleeding from the right ear. Four months later he complained of headache, and especially of dizziness on slight exertion. The dizziness continued for more than two years. Neurologic examination, made on June 3, showed the left pupil to be larger than the right, and impaired hearing on the right. Weber's test was referred to the right. His memory and calculating ability were poor. Roentgenograms of the skull showed a linear fracture of the right side running from the midvault to the external auditory meatus. The Wassermann reaction of the blood was negative.

On Jan. 25, 1927, the auditory test showed: bilateral and mixed conductive and receptive type of lesion, more marked on the right; the vestibular test: the right horizontal canal showed a subnormal response after turning; the right horizontal canal was the only one on either side to function after douching; a picture of an angle tumor.

Case 24 (C-26).—*Concussion; intracranial hemorrhage; neurosis.*—A man, aged 65, a fireman, was referred by an insurance carrier on March 16, 1926. On Jan. 5, 1926, he fell backward 5 feet, striking his head on a piece of iron. He was momentarily dazed. The following day diplopia developed, which persisted for three weeks. Eight days after the accident left peripheral facial paralysis developed, which subsequently cleared up. He complained of headache and dizziness, which persisted for more than four years. He also complained of deafness and tinnitus on the right side. There was no bleeding from either ear at the time of injury. Neurologic examination, made on March 16, revealed slight weakness of the left sixth and seventh cranial nerves. Hearing was impaired on the right. Tests with the tuning fork showed bone conduction to be better than air conduction on the right side, and air conduction better than bone conduction on the left. Weber's test was referred to the left. Roentgenograms of the skull were negative for fracture. The Wassermann reaction of the blood was negative. A posttraumatic neurosis developed, and the patient did not return to work.

TABLE 1.—*Results of E*

Case No.	Time Elapsed Between Injury and Examination	Auditory Impairment	Spontaneous Phenomena			Nystagmus: Rotatory		Nystagmus: Caloric			
			Nystagmus	Station	Past Pointing	After Turning to Right	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	After Douching Left Ear, Head Back
(1) County	4 mo.	Marked on left side, of inner ear type	Rotary to left especially with head back	Good	None	30 seconds' duration; poor amplitude	35 seconds' duration; poor amplitude	Appeared after 1 min. 45 sec.; faint	Appeared after 2 min. 10 sec.; faint	Perverted, i.e., vertical instead of horizontal	Good amplitude
(2) C-16	16 mo.	Moderate on right, of inner ear type	None	Slight general swaying	To right with both arms	12 seconds' duration; very poor amplitude	10 seconds' duration; very poor amplitude	None	None	None	Good amplitude
(3) C-35	3½ mo.	Moderate on both sides, of inner ear type	None	Slight general swaying	3 inches to right with left arm	12 seconds' duration; very poor amplitude	11½ seconds' duration; very poor amplitude	None	None	None	Poor amplitude
(4) C-48	10 mo.	Moderate on both sides, of mixed inner and middle ear type	None	Good	None	23 seconds' duration; good amplitude	20 seconds' duration; good amplitude	None	None	Poor	None
(5) C-38	5 wk.	Very slight on both sides, of inner ear type	None	Good	None	14 seconds' duration; fair amplitude	11 seconds' duration; fair amplitude	None	None	None	None
(6) C-0	5 mo.	Complete on right and moderate on left, of inner ear type	None	Fell toward right ear with head all positions	Slightly to right with both arms	5 seconds' duration; poor amplitude	6 seconds' duration; poor amplitude	None	None	None	Good amplitude
(7) C-60	10 mo.	Middle ear type on right plus moderate inner ear type on both sides	None	Good	None	22 seconds' duration; good amplitude	27 seconds' duration; good amplitude	Appeared after 40 sec.; good amplitude	Appeared after 40 sec.; good amplitude	Good amplitude	Good amplitude
(8) C-66	9 mo.	Unsatisfactory; marked on both sides, of inner ear type	None	Good	None	None	None	None	None	None	None
(9) ?-65	9 wk.	Marked middle ear type on right plus moderate inner ear type both sides	Small horizontal on looking to right	Good	None	14 seconds' duration; poor amplitude	20 seconds' duration; good amplitude	None	None	Good	Good amplitude
(10) C-64½	20 mo.	Moderate on both sides, of inner ear type	None	Good	None	21 seconds' duration; poor amplitude	22 seconds' duration; poor amplitude	None	None	Poor amplitude	Poor amplitude
(11) C-63	26 mo.	Slight in left ear, mixed middle and inner ear type	None	Good	To left with left arm	18 seconds' duration; poor amplitude	22½ seconds' duration; poor amplitude	Appeared after 25 sec.; hyperactive; good amplitude	Appeared after 25 sec.; hyperactive; good amplitude	Good amplitude	Good amplitude

	Past Pointing						
After Turning to Right	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	After Douching Left Ear, Head Back	Constitutional Symptoms	Summary
ssed in arm; points ft ad of t	Crossed in left arm; i.e., points to right instead of left	Within normal limits	Within normal limits	Within normal limits	Within normal limits	Slight from right ear; more from left ear	Spontaneous nystagmus; crossed past pointing in left arm; perverted nystagmus from right horizontal canal after douching
ssed in arm	Absent in left arm; crossed in right arm	Practically absent in both arms	Practically absent in both arms	Practically absent in both arms	Within normal limits	Absent except from horizontal canal	Neuro-otologic picture characteristic of angle tumor, except for auditory impairment which was on opposite side; basilar fracture
longed ght ; crossed ft arm	Crossed in right arm	Subnormal	Subnormal	Subnormal	Normal	Absent	Same as no. 2, except that there was no auditory impairment; slow on top of head; 4+ Wassermann reaction
ent in h arms	Absent in both arms	Subnormal	Practically absent in both arms	Practically absent in both arms	Practically absent in both arms	Absent	Practically same as nos. 2 and 3; no past pointing
?	?	Practically absent in both arms	Absent in both arms	?	Within normal limits	Absent	Steeple jack; poor cooperation; labyrinths nonresponsive to douching
rtically ent	Practically absent	Practically absent in both arms	Practically absent in both arms	Subnormal	Within normal limits	Absent	Same as nos. 2, 3 and 4; auditory and vestibular apparently out, except some past pointing and nystagmus after douching left horizontal canal
ssed in t arm	Shortened	Crossed in left arm	Absent in right arm	Normal	Absent in right arm	Slight	Abnormalities in past pointing of doubtful value
ent	Absent	Absent in both arms	Absent in both arms	Absent in both arms	Absent in both arms	Absent	Nonresponsive labyrinth on both sides
?	?	Subnormal	Absent in right arm	Absent in both arms	Absent in both arms	Present	
ithin nor- al limits	Crossed in left arm	Practically absent in both arms	Practically absent in both arms	Practically absent in both arms	Practically absent in both arms	Absent	Abnormalities in past pointing; no responses from vertical canals
ot done account f nausea	Not done on account of nausea	Practically absent i n right arm	Absent in both arms	Within normal limits	Wide with both arms	Marked	Hyperactive responses; abnormalities in past pointing in right arm after douching; marked constitutional symptoms

Case No.	Time Elapsed Between Injury and Examination	Auditory Impairment	Spontaneous Phenomena			Nystagmus					
						Rotatory		Caloric			
			Nystagmus	Station	Past Pointing	After Turning to Right	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	After Douching Left Ear, Head Back
(12) C-47	3 mo.	Marked on right, of middle ear type; perforated drum on this side	None	Good	None	30 seconds' duration; good amplitude	28 seconds' duration; good amplitude	Appeared after 1 min. 10 sec.; good amplitude	None	Perverted, i.e., oblique to left instead of horizontal	Good amplitude
(13) C-44	9 days	No impairment of inner ear function; slight on left, middle ear type	None	Good	None	26 seconds' duration; fair amplitude	26 seconds' duration; fair amplitude	Appeared after 35 sec.; good amplitude	Appeared after 30 sec.; good amplitude	Good amplitude	Good amplitude
(14) C-43	8 wk.	Marked on left, of inner ear type	None	?	To right with right arm; to left with left arm	21 seconds' duration; good amplitude	23 seconds' duration; good amplitude	None	Appeared after 1 min.; very slow	Good amplitude; extremely slow	Good amplitude; extremely slow
(15) C-42	12 mo.	Slight on right, of middle ear type; complete on left, of inner ear type	None	Good	None	23 seconds' duration; poor amplitude	23 seconds' duration; poor amplitude	Appeared after 30 sec.; good amplitude	Appeared after 30 sec.; poor amplitude	Good amplitude	Good amplitude
(16) C-39	30 days	None	None	Tended to fall backward with head on either shoulder	To left with both arms	26 seconds' duration; good amplitude	27 seconds' duration; good amplitude	Appeared after 1 min. 15 sec.; poor amplitude	None	Good amplitude	Poor amplitude
(17) C-21	2 yr.	?	Horizontal to both sides, greater to left	Fell back and to left	To left with right arm	30 seconds' duration; good amplitude	30 seconds' duration; good amplitude	None	Appeared after 45 sec.; good amplitude	Good amplitude	Good amplitude
(18) C-20	12 mo.	Slight on both sides, of inner ear type	None	None	None	20 seconds' duration; atypical due to paralysis of eye muscle	12 seconds' duration; atypical due to paralysis of eye muscle	Appeared after 30 sec.; good amplitude	Appeared after 35 sec.; good amplitude	Good amplitude	Good amplitude
(19) C-13	4½ mo.	Complete on left	?	?	?	19 seconds' duration; poor amplitude	15 seconds' duration; poor amplitude	None	Appeared after 30 sec.; fair amplitude	Poor amplitude	Good amplitude
(20) C-11	2 yr.	Complete on right side	Large horizontal to left on looking to left	?	None	Patient confined to his bed		None	Fair amplitude	None	Good amplitude
(21) C-9	8 mo.	None	Rotary to right on looking up	General swaying	None	16 seconds' duration; good amplitude, but slow	25 seconds' duration; good amplitude, rapid	Appeared after 30 sec.; good amplitude	Appeared after 55 sec.; good amplitude	?	?
(22) C-6	2 mo.	None	None	?	None	21 seconds' duration; fair amplitude	10 seconds' duration; poor amplitude	None	None	None	Good amplitude

Thirty Cases—Continued

Past Pointing							
After Turning to Right	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	After Douching Left Ear, Head Back	Constitutional Symptoms	Summary
ortened	Within normal limits	Absent in both arms	Absent in right arm	Absent in both arms	Absent in right arm	Absent from right ear; present from left ear	Absent past pointing in right arm; both ears
ithin normal limits	Shortened in right arm	?	Absent in right arm	Absent in left arm	Absent in right arm	Present; marked	Practically normal except for abnormalities in past pointing in right arm
ortened	Shortened	Absent in both arms	Absent in right arm	Absent in right arm; crossed in left arm	Normal	Absent	Right vertical canal nonfunctioning after douching; abnormal past pointing
hortened n left arm; bsent in ght arm	Crossed in right arm; shortened in left arm	Present	Present	Exaggerated in right arm	Practically absent in both arms	Absent	Marked auditory, slight vestibular impairment on left side; abnormality in absent past pointing from left horizontal canal after douching
bsent; i.e., oes not hange the pontaneous past ointing	Absent in right arm; prolonged in left arm	Absent in both arms	Absent in both arms	Absent in both arms	Absent in both arms	Absent	Practically no change in spontaneous past pointing even in presence of nystagmus and motion sensing
bsent	Absent in right arm	Absent	Absent	Absent	Absent	Absent	Right vertical nonresponsive to douching; practically no past pointing from other active canals; constitutional symptoms absent
bsent in eft arm	Crossed in right arm	Crossed in left arm	Crossed in right arm	Crossed in left arm	Absent in right arm	?	Active nystagmus; abnormal past pointing
?	?	Absent in left arm	Absent in right arm	Absent in right arm	Absent in right arm	Absent	
Patient confined to his bed		?	?	?	?	Absent	No response from right auditory or vestibular apparatus
Absent in left arm	Crossed in right arm; shortened in left arm	?	?	?	?	Marked	Tests not completed on account of vomiting; hyperactive responses; abnormalities in past pointing
?	?	Absent in both arms	Absent in both arms	Absent in both arms	Absent in both arms	Absent	Right side nonresponsive; left vertical canals out; angle tumor

TABLE 1.—*Results of Examina*

Case No.	Time Elapsed Between Injury and Examination	Auditory Impairment	Spontaneous Phenomena			Nystagmus					
						Rotatory		Caloric			
			Nystagmus	Station	Past Pointing	After Turning to Right	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	After Douching Left Ear, Head Back
(23) C-51	12 mo.	Moderate on right, of inner and middle ear type; same on left, but less marked	Horizontal to right on looking to right	Tended to fall toward right ear with head in all positions	To right with right arm	22 seconds' duration; good amplitude	17 seconds' duration; fair amplitude	None	None	Small amplitude	None
(24) C-26	4 mo.	Marked on right, of middle and inner ear type; middle ear type on left	Large horizontal to left	General swaying	Slight to left with left arm	14 seconds' duration; good amplitude	10 seconds' duration; fair amplitude	None	Appeared after 1 min. 15 sec.; fair amplitude	None	Good amplitude
(25) C-64	12 mo.	Moderate on left, of middle ear type	None	Good	To left with left arm	21 seconds' duration; good amplitude, slow	21 seconds' duration; good amplitude, slow	None	None	Fair amplitude	Good amplitude
(26) C-65½	4 mo.	Marked on both sides, of inner ear type	None	Good	None	34 seconds' duration; good amplitude	20 seconds' duration; fair amplitude	Appeared after 45 sec.; good amplitude	Appeared after 30 sec.; good amplitude	Good amplitude	Good amplitude
(27) C-66	6 mo.	Moderate on right, of middle ear type	Horizontal to right on looking to right	Fell toward left ear with head in all positions	To left with right arm	9 seconds' duration; poor amplitude	9 seconds' duration; poor amplitude	None	None	Good amplitude	None
(28) County	6 wk.	.....	None	Fell to right with head in all positions	None	35 seconds' duration; fair amplitude	36 seconds' duration; good amplitude	None	Appeared after 45 sec.; good amplitude	None	Good amplitude
(29) County	3 mo.	?	None	None	None	12 seconds' duration; poor amplitude	12 seconds' duration; poor amplitude	Appeared after 45 sec.	Appeared after 55 sec.; perverted in type, i.e., oblique to right instead of rotary to right	Good amplitude	Good amplitude
(30)	19 mo.	Complete on right; conductive type	Rotary to both sides, small	Fell to right	To right with right arm	7½ seconds' duration; very small amplitude, rapid	7½ seconds' duration; very small amplitude, rapid	None	None	None	Good amplitude

Thirty Cases—Continued

	Past Pointing						
After Turning to Right	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	After Douching Left Ear, Head Back	Constitutional Symptoms	Summary
Within normal limits	Shortened in right arm	Absent in both arms	Absent in both arms	Absent	Absent	Absent	Angle tumor picture, with exception of auditory picture
Practically absent in right arm; crossed in left arm	Shortened in both arms	Absent in both arms	Within normal limits	Absent	Within normal limits	Absent from right ear; present from left ear	Right side nonfunctioning
Staggered in right arm; practically absent in left arm	Absent in right arm; shortened in left arm	Absent in both arms	Subnormal	Subnormal	Practically absent	Present from left ear	Absent responses from vertical canals; abnormalities in past pointing
Not reliable	Not reliable	Absent in right arm	Absent in right arm	Crossed in left arm	Normal	Present	Hyperactive responses from left side; abnormalities in past pointing
Practically absent in right arm; crossed in left arm	Shortened in both arms	Practically absent in right arm	Practically absent in both arms	Absent in right arm; subnormal in left arm	Absent	Absent	No responses from left side; abnormalities in past pointing; angle tumor picture left, except for auditory impairment
Slightly shortened in both arms	Shortened in left arm	Crossed in both arms	Normal	Slightly subnormal	Absent	Present from right ear	No nystagmus after douching right ear; abnormalities in past pointing from left side with normal nystagmus
?	?	Subnormal	Subnormal	Crossed in right arm; absent in left arm	Within normal limits	Present	Perverted responses from left vertical canals; no skull fracture; Wassermann reaction negative (blood and spinal fluid); vertigo came on 4 days after accident; diagnosis: concussion
Shortened in both arms	Shortened in right arm; absent in left	Practically absent	Practically absent	Practically absent	Practically absent	Absent	Cerebellopontile angle syndrome without typical auditory observations

On July 15, 1927, the auditory test showed: receptive type of lesion on both sides, more marked on the right; the vestibular test: spontaneous, horizontal nystagmus to the left; all of the canals nonresponsive on the right side; hyperactive responses on the left side.

CASE 25 (C-64).—*Concussion; neurosis.*—A man, aged 65, a foreman, was referred by his employer on Aug. 25, 1927. On Aug. 18, 1926, he was struck on the left side of the head by a lumber carrier and was rendered unconscious for a few hours. There probably had been bleeding from the left ear. A year later he complained of occipital headache and dizziness. The dizziness was so marked that he was unable to ride in an elevator or on moving vehicles. He complained of constant tinnitus in the left ear, like the "ringing of bells." He also complained of deafness on the left side. Neurologic examination, made on Aug. 25, 1927 gave negative results, save for deafness of the left ear. Roentgenograms of the skull showed no fracture, but revealed a fracture of the jaw, as well as of the left transverse process of the seventh cervical vertebra.

On August 29, the auditory test showed: left-sided impairment, conductive type of lesion; the vestibular test: the vertical canals failed to respond to douching; past pointing practically absent.

CASE 26 (C-65½).—*Concussion; intracranial hemorrhage, left; right hemiplegia and aphasia.*—A man, aged 46, a laborer, was referred by Dr. W. T. Cade, Jr., on May 12, 1927. He was injured on April 7, 1927, when he was struck on the left side of the head by a guy rope under tension. He was unconscious for two hours and right-sided hemiplegia and aphasia developed. Neurologic examination, made on May 12, showed residual aphasia and right hemiparesis. Operation for removal of the clot was refused. Roentgenograms of the skull failed to reveal a fracture. The Wassermann reaction of the blood was negative. He was observed for a period of two years and ten months, during which time he did not return to work.

On Sept. 14, 1927, the auditory test showed: bilateral receptive type of lesion; the vestibular test: responses within normal limits, except for abnormalities in past pointing.

CASE 27 (C-66).—*Linear fracture of the left temporal region; concussion and contusion of the brain; left facial paralysis.*—A man, aged 46, a tree trimmer, was referred by Dr. W. M. Briggs on March 20, 1927. On March 10, 1927, he fell from a tree, striking the concrete. He was immediately rendered unconscious and remained so for four days. There was bleeding from the left ear, and on the following day left peripheral facial paralysis developed. He had been deaf in the right ear for twenty-five years previously, and there was an old perforation of the drum on this side. He complained of headaches and dizziness for more than a year following his accident. Neurologic examination, made on March 20, gave negative results, except for the left peripheral facial paralysis. There was bleeding from the left ear. The paralysis of the face cleared up in three months. A year later he still complained of headaches and dizziness. Roentgenograms of the skull showed a linear fracture in the left temporal region. The Wassermann reaction of the blood was negative.

On September 26, the auditory test showed: conductive type of lesion on the right; the vestibular test: responses absent from the left side; the horizontal canal functioned only on the right side.

CASE 28 (County).—*Concussion and contusion of the brain; fracture of the skull.*—A man, aged 35, a laborer, was admitted to the Los Angeles General Hospital on March 18, 1927. On Feb. 26, 1927, he was knocked down by a taxi, being

rendered unconscious and remaining so for twenty-four hours. Three weeks later, his principal symptoms were headaches and dizziness. Neurologic examination at this time showed unsteadiness of gait with a tendency to fall to the right, increase of the deep reflexes and a double positive ankle clonus, but otherwise it gave negative results. Roentgenograms of the skull showed a linear fracture in the right parietofrontal region.

On March 31, the auditory test was not done; the vestibular test showed: responses absent on the right side; normal on the left side; abnormalities in past pointing from both sides.

CASE 29 (County).—*Concussion* (?).—A woman, aged 25, a hospital attendant, was admitted to the Los Angeles General Hospital on Sept. 20, 1926. On Sept. 11, 1926, while riding on a roller coaster, she struck her head, but was not rendered unconscious. She worked for the next two days, when she was forced to stop because of headache and dizziness. Three months after the injury she still had headache and dizziness. Neurologic examination showed moderate internal squint of the right eye, greatly exaggerated deep reflexes, the reflexes being quicker on the right than on the left, and a suggestive Babinski sign on both sides, but otherwise gave negative results. Roentgenograms of the skull were negative for fracture. The Wassermann reaction of the blood was negative. An examination of the spinal fluid made three weeks after the injury, showed a clear fluid under increased tension, but otherwise gave normal results.

On December 9, the auditory test was not done; the vestibular test showed: perverted nystagmus from the left vertical canals; abnormalities in past pointing.

CASE 30 (C-30).—*Probable concussion.*—The patient was first seen nineteen months after an injury to the skull, complaing of deafness and vertigo.

The auditory test showed: complete deafness on the right side, a conductive type of lesion; the vestibular test: spontaneous rotatory nystagmus to each side; spontaneous falling to the right; spontaneous past pointing to the right; typical vestibulo-auditory picture of cerebellopontile angle lesion; i. e., the horizontal canal was the only one functioning on the left side.

RÉSUMÉ OF RECHECKED CASE REPORTS

CASE 1 (C-3).—*Concussion.*—A man, aged 61, a laborer, who was referred by Dr. John D. Gillis, was injured on March 18, 1925, when he fell 5 feet. He was rendered unconscious, and remained so for a few hours. There was an abrasion back of the left mastoid region. No bleeding from the ears was mentioned. Six months later his principal complaints were headache and dizziness. Neurologic examination, made on September 10, gave essentially negative results. Roentgenograms of the skull were negative for fracture. The dizziness was as marked a year after the injury as at any time during the year.

On Sept. 10, 1925, Dec. 4, 1925, March 17, 1926, Aug. 3, 1926, Nov. 17, 1926, and April 6, 1927, the auditory test showed: complete deafness on the left side; the vestibular test: delayed responses from the left horizontal canal; delayed nystagmus from the left vertical canals and perverted nystagmus from the left horizontal canal after douching; abnormalities in past pointing. On reexaminations extending over a period of a year, the responses from the left set of canals was found to have disappeared.

CASE 2 (C-50).—*Concussion.*—A man, aged 48, a grader, was referred by Dr. W. T. Cade, Jr., on Jan. 10, 1927. He was injured in an automobile collision on Nov. 27, 1926, when he was rendered unconscious for a few minutes. He received a laceration of the right temple, but there was no bleeding from either ear. At

TABLE 2.—*Results of E*

Case No.	Time Elapsed Between Injury and Examination	Auditory Impairment	Spontaneous Phenomena			Nystagmus					
						Rotatory		Caloric			
			Nystagmus	Station	Past Pointing	After Turning to Right	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	
1 C-3	(Dias) 6 mo.	Complete on left side, of middle and inner ear type; marked on right side, of middle and inner ear type	None	Fell to left	To right with right arm (3 in.); to left with left arm (5 in.)	25 seconds' duration; fair amplitude	16½ seconds' duration; fair amplitude	Appeared after 45 sec.; good amplitude	Appeared after 2 min.; very poor	Good amplitude	
	9 mo.	Bone conduction returning on right side indicating some return in inner ear function; still absent in left ear	None	Fell to left	To right with right arm (3 in.); to left with left arm (4 in.)	15 seconds' duration; fair amplitude	15 seconds' duration; fair to good amplitude	Good amplitude	Absent	Good amplitude	
	1 yr.	About the same as last visit	None	Fell to left	Same as when last examined	14 seconds' duration; fair amplitude	16 seconds' duration; fair to good amplitude	Appeared after 40 sec.; good amplitude	Absent	Good amplitude	
2 C-50	(Southard) 1 yr. 2 mo.	Moderate on both sides, inner ear type; worse on right	Slight rotary to the left	Moderate swaying	To right with left arm (3 in.)	38 seconds' duration; good amplitude	40 seconds' duration; good amplitude	Appeared after 1 min. 30 sec.; good amplitude	Appeared after 35 sec.; good amplitude	Fair amplitude	
	1 yr. 5 mo.	No change since last visit	None	Moderate swaying	Practically none	28 seconds' duration; poor amplitude	35 seconds' duration; fair amplitude	Appeared after 2 min. 26 sec.; very poor; practically absent	Appeared after 1 min.; very poor; practically absent	Very small	
	1 yr. 7 mo.	No change since last visit	None	General swaying	None	32 seconds' duration; fair amplitude	38 seconds' duration; fair amplitude	Appeared after 2 min.; fair amplitude	Appeared after 51 sec.; fair amplitude	Good amplitude	
3 C-59	(Saueressig) 1 mo.	Mixed middle and inner ear type, approximately equal on the two sides	Horizontal to right and left, respectively	Tendency to fall forward	To left with both arms	27 seconds' duration; good amplitude	28 seconds' duration; good amplitude	Appeared after 20 sec.; good amplitude	Appeared after 60 sec.; good amplitude	Good amplitude	
	6 mo.	Rather marked decrease with tuning forks	Doubtful	Fairly good	Practically absent	22 seconds' duration; small amplitude	29 seconds' duration; good amplitude	Absent	Absent	Very small	
4 C-56	(Littlefield) 3 mo.	No perception of tuning forks on left; bone conduction slightly shortened on right	None	Good	Doubtful	27 seconds' duration; good amplitude	17 seconds' duration; good amplitude	Appeared after 45 sec.; good amplitude	Appeared after 2 min. 30 sec.; good amplitude	Good amplitude	
	6 mo.	Marked decrease in inner ear function on right side; Continued absence of function on left	Doubtful	Good	Doubtful	12 seconds' duration; poor amplitude	16½ seconds' duration; poor amplitude	Appeared after 60 sec.; poor amplitude	Appeared after 60 sec.; poor amplitude	Good amplitude	

	Past Pointing						
...fter ...rning to ...ight	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	After Douching Left Ear, Head Back	Constitutional Symptoms	Summary
?	?	Normal in right arm; absent in left arm	Absent in right arm; normal in left arm	Somewhat exaggerated in both arms	Within normal limits	Absent	Bilateral auditory impairment apparently complete on left side, of inner ear type; left vestibular organ gave no nystagmus from vertical canals; perverted nystagmus from horizontal canals; responses somewhat exaggerated from right ear
...mal in ...t arm; ...nt in ...arm	Within normal limits	Normal in right arm; subnormal in left arm	Absent in both arms	Within normal limits	Absent	Absent	Some return of auditory function on right
...gger- ...d in ...t arm; ...sed in ...arm	Crossed in left arm	Normal in right arm; absent in left arm	Absent in both arms	Absent in left arm	Absent	Absent	Responses were all practically the same as when examined 3 months previously; staggering, vertigo and deafness continued the same
...y ex- ...erated	Very exaggerated	Subnormal in right arm	Subnormal	Absent in right arm	Exaggerated in left arm	Moderate on right	Spontaneous nystagmus with hyperactive responses on left side
...htly ex- ...erated	Within normal limits	Absent	Absent	Within normal limits	Within normal limits	Slight	Spontaneous nystagmus gone; irritability of vertical canal; responses diminishing
...olonged; ...ssed in ...t arm	Within normal limits	Subnormal	Subnormal	Within normal limits	Within normal limits	Slight	No change from last examination; picture changed from hyperirritability to hypo-irritability
...thin normal ...l limits	Within normal limits	Subnormal	Not taken	Practically absent	Not taken	Marked from both sides	Moderate hyperactive responses, especially on right side in presence of absent past pointing
...thin normal ...l limits	Within normal limits	Practically absent	Practically absent	Absent	Absent	Absent	Marked decrease in auditory function over period of six months; nystagmus hyperactive at first, responses markedly hypo-active after six months
...ortened ...right ...m; absent ...left arm	Within normal limits	Subnormal	Practically absent	Practically absent	Crossed in both arms	Absent	Auditory function impaired on left; vestibular function impaired on same side; abnormalities in past pointing
...ach ...ortened ...both ...ms	Prolonged in both arms	Absent	Absent	Absent	Absent	Absent	In presence of marked improvement in subjective symptoms, i. e., decrease in vertigo, the patient showed a marked decrease in auditory and vestibular function

TABLE 2.—*Results of Examina*

Case No.	Time Elapsed Between Injury and Examination	Auditory Impairment	Spontaneous Phenomena: Nystagmus	Spontaneous Phenomena: Station	Spontaneous Phenomena: Past Pointing	Nystagmus: Rotatory: After Turning to Right	Nystagmus: Rotatory: After Turning to Left	Nystagmus: Caloric: After Douching Right Ear, Head Up	Nystagmus: Caloric: After Douching Left Ear, Head Up	Nystagmus: Caloric: After Douching Right Ear, Head Back	Nystagmus: Caloric: After Douching Left Ear, Head Back
5 C-67	(Lyle) 2 mo.	Mixed middle and inner ear type on right, moderate	None	Good	None	14 seconds' duration; good amplitude	16 seconds' duration; good amplitude	Appeared after 48 sec.; fair amplitude	Appeared after 15 sec.; fair amplitude	Good amplitude	Good amplitude
	5 mo.	Improved on right side	None	Good	None	15½ seconds' duration; good amplitude	15 seconds' duration; good amplitude	Practically absent	Appeared after 1 min. 10 sec.; small amplitude	Good amplitude	Good amplitude
6 C-2	(Seagraves) 4 mo.	Complete, of inner ear type on right; moderate on left of middle and inner ear type	None	?	To right with right arm (4 in.); touched with the left arm	19 seconds' duration; good amplitude	11½ seconds' duration; fair amplitude	Absent	Appeared after 2 min.; very small amplitude	Absent	Good amplitude
	1 yr. 3 mo.	Tuning fork tests indicate no improvement; heard spoken voice better on both sides	None	?	To right with right arm (2 in.); to right with left arm (2 in.)	18 seconds' duration; good amplitude	18 seconds' duration; good amplitude	Absent	Appeared at end of 1 min. 30 sec.; good amplitude	Absent	Good amplitude
	1 yr. 9 mo.	Marked impairment in left to spoken voice; moderate impairment on the right side	None	None	None	18 seconds' duration; good amplitude	15 seconds' duration; good amplitude	Absent	Appeared at end of 1 min. 30 sec.; good amplitude	Absent	Good amplitude

the end of two weeks, he tried to work, but was obliged to stop because of dizziness. The dizziness increased during the next five months. At times he was nauseated and would vomit, and during his attacks of vertigo he staggered toward the right. Neurologic examination revealed the right pupil to be larger than the left; the Romberg sign was positive; his gait was unsteady and he reeled toward the right; otherwise the results were negative. Roentgenograms of the skull showed no fracture. The Wassermann reaction of the blood was negative.

On January 12 and on March 9, the auditory test showed: moderate impairment, bilateral, receptive lesion, more marked on the right side; the vestibular test: slight spontaneous nystagmus to the left; hyperactive responses from the horizontal canals after turning; hyperactive responses from the left labyrinth after douching; abnormalities in past pointing. Reexamination five months later showed delayed responses from the right vertical canals after douching. The picture had changed from one of hyperirritability to one of hypo-irritability.

CASE 3 (C-59).—*Concussion.*—A man, aged 71, a carpenter, was referred by Dr. W. T. Cade, Jr., on June 27, 1927. On March 21, 1927, he was struck on the head by a piece of falling timber. He was not rendered unconscious, but stated that there was a terrific noise in his head. There was no bleeding from either ear. Two days later he complained of headache and dizziness, which continued for many months. He had a violent ringing in the right ear and a screeching sound in the left. For many years he had been "hard of hearing." Neuro-

Cases Rechecked—Continued

	Past Pointing						
After Turning to Right	After Turning to Left	After Douching Right Ear, Head Up	After Douching Left Ear, Head Up	After Douching Right Ear, Head Back	After Douching Left Ear, Head Back	Constitutional Symptoms	Summary
ortened both ms	Shortened in both arms	Absent	Absent	Absent	Absent	Absent	Abnormalities in past pointing in presence of active nystagmus
hortened right rm; absent left arm	Absent in in both arms	Absent	Absent	Absent	Absent	Absent	Subjectively improved in regards to vertigo and hearing; objectively, some improvement in hearing on right side; past pointing absent from the first; nystagmus after douching hyperactive on right at first, disappeared on second examination
?	?	Absent in both arms	Within normal limits (3 and 3)	Absent in both arms	Within normal limits	Absent from right side; slight from left side	Right vestibular and cochlear impairment
lightly abnormal n both rms	Shortened in right arm; absent in left arm	Practically absent in both arms	Absent in right arm; subnormal in left arm	Crossed in both arms	Absent in left arm; crossed in right arm	.....	Slight improvement in auditory function on both sides; right vestibular apparatus still failed to respond
Within normal limits	Within normal limits	Absent in both arms	?	Absent in both arms	Within normal limits	Absent	Five months later showed practically same picture in regard to vestibular function; subjectively much improved; had had no attacks of vertigo for three weeks prior to last examination

logic examination gave negative results. The patient showed advanced senile changes incident to his age. Roentgenograms of the skull showed no fracture. The Wassermann reaction of the blood was negative. For a period of fourteen months his symptoms continued unabated, and he was totally incapacitated.

On Aug. 18, 1927, Nov. 7, 1927, and July 7, 1928, the auditory test showed: bilateral impairment, mixed conductive and receptive type of lesion; the vestibular test: when first examined, vestibular responses were hyperactive, with spontaneous, horizontal nystagmus to the right; five months later they were hypo-active, i. e., no nystagmus from the vertical canals after douching. Past pointing had disappeared at a subsequent examination.

Case 4 (C-56).—*Concussion and contusion of the brain; linear fracture of the left temporal bone.*—A man, aged 32, a laborer, was referred by Dr. George W. Jones on July 10, 1928. On Feb. 22, 1927, he fell 15 feet, striking the left side of his head on some brick. He was rendered unconscious, remaining so for about two weeks. There was profuse bleeding from the left ear. Six months later he complained of deafness in the left ear, ringing in the left ear, occasional headache, dizziness, forgetfulness, confusion and irritability. Neurologic examination at this time showed irregular pupils, impairment of hearing in the left ear and a fine tremor of both hands in attempting coordination tests, but otherwise gave negative results. Roentgenograms of the skull revealed a linear fracture in the left temporal region.

On May 20 and October 21, the auditory test showed: no perception of sound on the left side; the vestibular test: reduced responses from the right horizontal canal after turning; abnormalities in past pointing throughout; responses from the left vertical canals much delayed after douching. The vestibular reactions were much more hypo-active on reexamination six months later.

CASE 5 (C-67).—*Concussion; basal fracture of the skull.*—A man, aged 24, a lather, was referred by Dr. J. Rollin French on Oct. 17, 1927. He was injured on Aug. 18, 1927, when his motorcycle collided with a truck. He was rendered unconscious for ten minutes, and there was bleeding from the right ear. Bell's palsy subsequently developed on the right side. Two months after the injury he complained of faintness and dizzy spells. The dizziness developed about two weeks after the accident, and was accentuated by getting up suddenly or putting the head backward. The patient also complained of constant tinnitus in the right ear, which he described as sounding like "a leak in an air line."

On examination, slight weakness of the right side of the face persisted, although Bell's palsy had practically cleared up. There were a horizontal nystagmus with the quick component to the left when the patient looked toward the left, impairment of hearing on the right with tinnitus in this ear and increased irritability. Roentgenograms of the skull showed a linear fracture in the right parietal region extending into the mastoid cells. The Wassermann reaction of the blood was negative.

On Oct. 17, 1927, and Jan. 20, 1928, the auditory test showed: mixed conductive and receptive type of lesion on the right side; the vestibular test: good nystagmus after douching of the right ear, with absent past pointing; hyperactive nystagmus and constitutional symptoms after douching of the left ear, with almost absent past pointing. A second examination three months later showed the picture to be practically the same, except that the vertical canals on the right side were practically nonresponsive to douching.

CASE 6 (C-2).—*Concussion and contusion of the brain; linear fracture of both parietal bones; syphilis.*—A man, aged 29, a welder's helper, was referred by Dr. W. T. Cade, Jr., on Aug. 21, 1925. On April 21, 1925, he fell 18 feet from a ladder into a tank. He was rendered unconscious, recovering consciousness four hours later. Four months later he complained of constant ringing in the right ear, deafness of the right ear, irritability and deafness. Headache was not marked. There was a previous history of deafness of the left ear. Neurologic examination revealed inequality of the pupils, the right being larger than the left. There was unsteadiness of the eyes on looking to the right and the left. The patient was very deaf in both ears. He swayed in Romberg's position, and at times staggered. The knee reflexes were gone. Roentgenograms of the skull showed a linear fracture in each parietal bone, running toward the external auditory meatus. The Wassermann reaction of the blood was 4 plus. After three years of intensive antisyphilitic treatment, the Wassermann reaction of the blood was 1 plus. His hearing had not improved, although the dizziness was considerably less. He was unable to work.

On Oct. 20, 1926, and March 2, 1927, the auditory test showed: impairment on the right side, conductive type of lesion; vestibular: absent responses from the right side.

CONCLUSIONS

1. In practically all cases of cerebral concussion the patient complains of some form of equilibratory disturbance.

2. The disturbance of equilibration may be demonstrated by some abnormality in the neuro-otological observations.

3. These observations are usually indicative of mixed central and end-organ damage rather than involvement of the end-organ alone.

4. The most constant observations are abnormalities in the past pointing reactions.

5. Traumatic equilibratory disturbances arising in the end-organ are more apt to readjust themselves than are those of central origin.

6. Postconcussional vertigo should not be dismissed as psychogenic in origin until it has been checked by neuro-otological tests.

7. Postconcussional vestibular tests may entirely, or partially, simulate those found in the syndrome of tumor of the cerebellopontile angle.

A NEW TEST FOR BORROWED BONE CONDUCTION *

HENRY S. WIEDER, M.D.
PHILADELPHIA

In the study of labyrinthine deafness with the tuning forks, I have been confronted several times with the following observations: The patient gives a history of a sudden attack of nausea and vomiting accompanied by intense vertigo and total deafness in one ear. Otitis media and mastoiditis may or may not be present. Spontaneous nystagmus and past pointing may be present if the patient is seen soon enough after the onset of the attack.

Objectively there is a total loss of air conduction in one ear, as shown by all tests. When tested for bone conduction by the Weber test, the patient frequently refers the sound to the unaffected ear. When the tuning fork is placed over the affected mastoid, however, the patient frequently claims that he perceives the sound with the affected ear. This fact would rule out a dead ear. It is well known that it is difficult to localize sound definitely to one ear, especially when one is not cognizant of the fact that a sound produced at one spot on the skull can be carried to a distant spot by bone conduction. The following simple procedures, by varying the intensities of the sound produced, have helped me to clear up some doubtful cases.

Perform a Weber test; frequently the patient localizes the sound in the well ear. Place the 256 double vibration fork over the mastoid of the suspected side; occasionally the patient says that he hears the note on the affected side. In order to test whether or not this conduction is borrowed, create an artificial total obstruction on the affected side by closing the canal with the finger; if one is dealing with labyrinthine deafness and a dead ear, the patient does not notice any difference in the intensity of the sound, but if some hearing remains on that side, the sound is greatly increased.

In order to check up on this observation, remove the obstruction from the affected canal, and close the canal of the unaffected ear with the finger. The patient immediately notices a change in the intensity of the sound, and sometimes he even says that he hears the sound in the unaffected ear. Place the fork over the well mastoid; if the patient hears the sound with the unaffected ear, close the external auditory canal of the affected ear with the finger. The patient should notice no difference in the sound, whereas the closing of the canal of the unaffected ear magnifies the sound greatly.

---

* Submitted for publication, Dec. 30, 1930.

* Read before the Philadelphia Laryngological Society, Feb. 3, 1931.

With the results stated, one can be practically certain that despite the patient's statements to the contrary, all the sounds carried by bone conduction are being heard by the unaffected ear. Of course, confirmatory Bárány tests would help to establish the diagnosis. The procedure outlined should be of especial value in central deafness with involvement of the auditory nucleus or of the cochlear portion of the eighth nerve when the vestibular apparatus is unaffected.

2106 Spruce Street.

OTOSCLEROSIS

STAINING AND CHEMICAL REACTIONS *

E. W. HAGENS, M.D.
CHICAGO

The peculiar staining reactions of the foci in otosclerosis have long attracted attention. Various ideas have been expressed to explain the striking bluish-red appearance such as occurs with the hematoxylin-eosin stain. A number of investigators have attributed the bluish reaction to the presence of calcium. It has been supposed that the focus has so great a content of calcium that even after decalcification some of it would still be present and would show on staining with hematoxylin. The blue staining has also been thought to be due to the peculiar chemical characteristics of the ground or cement substance in the foci. Just what the chemistry of this is does not appear to be known. Another view is that the increased supply of blood and lymph to the focus produces the blue stain. It is also believed that the pink-staining tissue usually found just underneath the osteoblasts that line the enlarged marrow spaces is new bone. This bone takes a pink stain and not the blue of the hematoxylin. On the basis of increased calcium being present in the foci, Bruhl fed guinea-pigs madder (alizarin), hoping to produce a redness of the promontory which could be seen through the drum membrane. The results were negative, possibly owing to the lack of intensity of the color reaction. It is noted by many that certain foci do not stain deeply with hematoxylin, but are more reddish. These foci are believed to be of much longer duration. Other stains have been used to characterize the foci, such as van Gieson's, iron hematoxylin and carmine, but explanations as to the underlying cause of the atypical staining reaction have mainly regarded hematoxylin and eosin.

During the course of study of sections of otosclerosis of four temporal bones,[1] I became interested in the possibility of learning more about the staining and chemical reactions of the foci. The material at hand consisted of four bones showing otosclerosis.

1. Bone T was from a woman, aged 37, who had no previous history of aural infection, but who died following an acute infection of the temporal bone, with intracranial complications. The affected ear (the other ear was not obtained) revealed a focus in the "site of predilection."

---

* Submitted for publication, Jan. 10, 1931.

* Presented as candidate's thesis to the American Laryngological, Rhinological and Otological Society.

1. Hagens, E. W.: Otosclerosis, Arch. Otolaryng. **12**:273 (Sept.) 1930.

2 and 3. Bone Fe (right) and Bone Fe (left) were from a woman, aged 20, who had had a chronic discharge from the left ear for eighteen years following scarlet fever. Only slight deafness had been noted in this ear, and the other ear presumably was normal. The patient died following an acute suppuration in the left ear, which was associated with intracranial complications. On sectioning, both ears showed multiple foci of otosclerosis, although no such diagnosis had been made during life.

4. Bone W (left) was from a man, aged 70, who had been deaf for years. The patient thought that the condition was catarrhal. Dr. Shambaugh made a diagnosis of otosclerosis seven years before death. The sections showed a focus at the "site of predilection."

While experimenting on these sections, I found it advisable to use sections of ears not showing otosclerosis from other adult human beings, also sections from infants' ears and pigs' ears and other sections of bone. The results of the tests seemed sufficiently interesting to warrant reporting. The carrying out of the tests was made possible by the suggestions and aid of Dr. R. Bensley and Dr. G. W. Bartelmez of the University of Chicago.

In the first place, the foci from each of the four bones did not react in the same way. The differences, however, were a matter of degree or lack of reaction. Thus, one focus took the stain strongly, another weakly, while the last two did not show the stain. Just why these results obtained is a question. Difference in technic can hardly be the sole cause, because Fe (right) and Fe (left) showed these differences strikingly and they were from the same patient and were treated alike at all times. The possibility that the age of the focus influenced the staining and chemical reactions is, therefore, to be considered. A greater abundance of otosclerotic material will probably tend to clear up these matters in the future.

EXPERIMENTAL DATA

The sections were stained with a yellow aqueous solution of crystalline hematoxylin. Hematoxylin becomes blue only when it is mordanted with the salts of the heavy metals. The foci in Fe (right) stained a definite blue; the focus in T took a faint, but definite blue, whereas the foci of Fe (left) and W were negative. The stain was taken up by the bony structure of the foci, including the bone cells. The marrow spaces with their contained vessels and cells were not stained. The placing of the sections in the stain for twenty-four hours gave the same results. Sections of other temporal bones and long bones were negative. Because of the positive results in the foci of two of the bones, the presence of calcium or of some metallic salt seemed indicated.

Therefore, tests were made with alizarin and purpurin, both of which are used to reveal calcium. The results, however, did not confirm the suspicion that the sections contained calcium. Sections of the four bones, as well as of other temporal bones, and sections of long bones gave negative results. In several sections the foci were more prominent than the surrounding tissue, but close examination showed that this was due to the increased staining reaction of the marrow cells, while the bony framework of the foci was similar to the adjacent bone. When the sections were stained with alizarin for twenty-four hours, the aforementioned results were not altered. With these observations, other causes than calcium were sought.

The sections were treated with ammonium sulphide. Sections of Fe (right) showed a definite green reaction in the foci, while the other sections were entirely negative. The tests were repeated a number of times, but the same results were always obtained. This chemical reaction indicated the presence of iron in the foci of Fe (right). Confirmation of the reaction was carried out by treating the sections with an acid solution of potassium ferrocyanide. Sections so treated showed the definite blue reaction of ferric ferrocyanide or prussian blue in the foci. The foci of Fe (right) showed definitely; in T the reaction was slightly positive, while Fe (left) and W were entirely negative. Thus, the foci of the same two bones that were positive to hematoxylin were positive to the test for iron. It was noted that the distribution of the reaction, that is, the presence of the reaction in the bony framework of the foci and its absence in the marrow spaces and cells, was the same in both the hematoxylin and the iron tests. On reexamination of the sections stained with hematoxylin-eosin, it was seen that the iron-containing foci gave the deeper blue with hematoxylin-eosin.

Modifications of the test for iron were carried out for further information. Treating the sections with potassium ferrocyanide for twenty-four hours instead of for five minutes, in the case of Fe (left) and W, which had not stained with the usual method, showed that the sections as a whole had adsorbed colloidal prussian blue and that the foci were not any more deeply stained than the rest of the tissue. Next, sections of Fe (right) and Fe (left) for control were kept in iron alum for forty-five minutes, thoroughly washed and then treated with potassium ferrocyanide. The sections showed blue as a whole, but the foci were still more blue. This indicated a greater capacity of adsorption of the bone in the foci. Other sections of Fe (left) were then subjected to the iron alum, after which they were placed in 0.5 per cent nitric acid solution, washed and, following this, were treated with potassium ferrocyanide. The results showed the foci still giving the blue reaction, but the acid had removed most of the iron from the rest of the tissue. This indicated an increased capacity of the foci for holding iron. Sections

of Fe (right) were treated for one hour with 0.5 per cent nitric acid and then treated with potassium ferrocyanide. The foci reacted just as when they had not been treated with acid, showing the inability of the acid to remove the iron from the foci in this length of time.

An ear (cochlea) from a full term infant was treated with potassium ferrocyanide, and the prussian blue reaction was obtained in the nuclei of the labyrinth (organ of Corti, especially) and at the edges of the bone tissue bordering the marrow spaces in the enchondral bony labyrinth. In the new-born infant, the marrow spaces are prominent and easily seen. Counterstaining with thiazine red was carried out to define further the site of the prussian blue reaction and confirmed the aforementioned observations. A bone from a finger of another new-born infant was also treated, and the blue reaction was noted in the bone of the diaphysis close to the cartilaginous junction. Sections of a normal temporal bone from an adult, treated as previously mentioned, gave no reaction.

Staining with toluidine blue was carried out on the otosclerotic sections, and the foci of Fe (right), Fe (left) and W were negative for a metachromatic reaction. However, in the sections of T, the focus stained a reddish-purple, especially in the anterior part, which was bluest with hematoxylin-eosin and slightly positive with pure hematoxylin and with the test for iron. In all the sections stained with toluidine blue the remnants of cartilage stood out prominently as purplish-red areas.

In order to test the possible action of the alum mordant in the usual hematoxylin-eosin stain, sections of bone T were placed in aluminum ammonium sulphate (alum) for forty-eight hours. Then the sections were stained with hematein. The result showed the focus taking a definite deep blue stain; previously, with pure hematoxylin the focus had stained only slightly better than the surrounding bone. Sections of bone Fe (left), which previously were entirely negative to pure hematoxylin alone, also showed the foci taking a deeper blue stain under this treatment. This reaction following the alum was interpreted to mean that the focus adsorbed colloidal aluminum hydroxide which, in turn, took up the hematein.

COMMENT

From the foregoing experiments several interesting observations are presented. It could not be proved whether calcium, as such, was present or absent in the foci. Apparently there is no chemical test by which calcium alone can be accurately demonstrated. So far as the tests with alizarin and purpurin were concerned, the reactions were negative. The ammonium sulphide test and the prussian blue reaction proved definitely the presence of iron in bone Fe (right) and T. Thus, the blue reaction in the foci of otosclerosis stained with hematoxylin-eosin may be due

to iron. The test in which mordanting with alum was followed by staining with hematein indicated that the affinity of the focus for the colloidal aluminum hydroxide caused the adsorption of the hematoxylin. Thus the focus may take a deep blue stain. This would explain the blue reaction with hematoxylin-eosin seen in the foci of Fe (left) and W, neither of which gave a reaction for iron. In general, it seems that either (1) the presence of iron in a focus or (2) the adsorption of hematoxylin through the medium of the colloidal aluminum hydroxide would account for the peculiar blue reaction with hematoxylin-eosin. Also, both factors may be present in a given focus, one supplementing the other. Thus it is seen that the foci of otosclerosis have a definitely increased adsorptive power for the iron and the aluminum hydroxide which is not possessed by the rest of the bony labyrinth.

The source of the iron in the focus is of interest. It may occur (1) antemortem or (2) postmortem. In the first instance, the pathologic process may have been such that an excess of iron was deposited there. The foci are usually vascular, and it is possible that iron from the blood had been freed and left in this region. It is known, of course, that the iron in the blood cells, as such, cannot be detected. Iron from the tissue cells may also have been freed by the destructive process and may furnish a source for the iron. If the iron was a postmortem affair, it arrived at the focus during the process of preparing the bone for sectioning. The necessity for determining complete decalcification furnishes a likely source for some of the iron. In the future, temporal bones will be run through without the use of the needle test, the x-rays having been found a more satisfactory guide; hence, this source for the iron will be removed and will not need to be considered. Another possibility is that the organically combined iron in the tissues may have been freed by the treatment with acid and adsorbed into the focus. Which of the aforementioned views is correct, it is difficult to say; perhaps several of the factors may be acting together.

The reactions to toluidine blue are of interest, but they are not chemically exact enough to draw conclusions.

I feel that microchemical tests may open an avenue of approach to otosclerosis that may have far-reaching possibilities. Limited material and the difficulty of getting it in a good state of preservation handicap work along this line. Chemical tests that are absolutely positive are much better guides than reactions that are indecisive and that lead to irrational speculation and conclusions.

30 North Michigan Avenue.

DELAYED HEALING FOLLOWING THE SIMPLE OPERATION ON THE MASTOID*

WILLIAM B. CHAMBERLIN, M.D.
CLEVELAND

In calling attention to delayed healing following the simple operation on the mastoid, I expect to play on a sympathetic cord—that of companions in misery. For to me, at least, the experience in treating some of the patients has been trying in the extreme. When I turned to the literature for help, or at least consolation, I was struck, not with the scarcity of articles on the subject, but with their almost entire absence.

Although all authors mentioned delayed healing following measles, diphtheria or scarlet fever, Kerrison alone referred, somewhat in extenso, to delayed healing in mastoiditis caused by the usual infecting organisms. I quote the following paragraph from his recent textbook under the title of "Arrested Repair":

> A post-operative condition, of which I have seen no mention in text books, but with which the surgeon has occasionally to deal, is characterized by the abortive type of granulations which line the bone cavity. Post-operative repair seems at first to follow a perfectly normal and favorable course. The bone cavity is soon lined by a layer of firm and apparently healthy granulations. The tympanic condition shows progressive improvement, and within a reasonably short period the drum membrane heals. In the post-auricular wound, however, the process of repair is arrested. There is little or no pus, and in fact no more secretion than is inseparable from a granulating wound. The granulations themselves, though of healthy appearance, are stationary, i. e., the process of tissue building seems arrested. Do what we may to increase the local blood supply and thereby stimulate a normal growth of new tissue, the aditus remains open and the post-auricular wound persists as a cavity of very considerable size. In my experience this condition—i. e., arrested repair, without evidences of osseous necrosis—is almost invariably an expression of anaemia or of some otherwise lowered constitutional state.

While he did not mention specific methods of after-treatment or efforts to stimulate repair of the wound, Kerrison suggested two possible solutions: (1) allowing the edges of the skin to grow into and so ultimately line the cavity of the wound, with much resulting disfigurement, and (2) the performing of a plastic operation.

TECHNIC OF OPERATION

Before proceeding with the subject in question, I should like to mention briefly the method that my associates and I follow at the present

* Submitted for publication, Jan. 28, 1931.
* Presented at the Middle Section Meeting, American Laryngological, Rhinological and Otological Society, Ann Arbor, Mich., January, 1931.

time in our clinic in the performance and after-treatment of the simple operation on the mastoid.

After the usual postauricular incision and removal of the outer cortex, the mastoid process is thoroughly exenterated until firm cancellous bone is encountered. The dura and sinus are freely uncovered, if the overlying bone is found to be involved. The zygomatic cells are uncapped and drained, and the whole mastoid cavity is thoroughly explored with a bent probe in order that no single cell be overlooked. The angle between the horizontal and the vertical portions above the wall of the sinus receives especial attention, owing to the possibility of overlooking cells in this locality. The antrum is freely opened, its outer wall being completely removed. Great care is taken that no chips of bone find lodgment in the aditus, thereby obstructing drainage from the middle ear. On the completion of the operation, the upper two thirds or three fourths of the wound are completely closed with Michel clamps, only a small opening being left inferiorly for the insertion of a rubber-covered cigaret drain. An end of this drain is carried directly into the antrum. No effort is made to fill the entire mastoid cavity with the drain. A loose wick is placed in the canal, the end in contact with the tympanic membrane.

The wound is dressed the day following the operation, the wick from the canal is removed, and the canal is thoroughly cleansed. On the fourth day, the skin clips and cigaret drain are removed. In case the middle ear is still discharging, the cigaret drain is replaced and the posterior wound kept open until the middle ear is dry.

The almost complete closure of the postauricular incision is a decided departure from our earlier procedure, in which the wound was packed almost completely open with iodoform gauze. We are convinced, however, that our present practice possesses the following marked advantages: 1. The dressing, i. e., the removal and replacement of the drain, is practically painless, as granulations cannot adhere to or grow into the rubber drain. 2. There is an almost complete absence of subsequent deformity; as a rule, only a fine linear scar with no depression remains. 3. There is a marked reduction in the time of convalescence, i. e., healing, with complete cessation of discharge from the middle ear and the wound in the mastoid.

The foregoing method has borne the test of long and favorable experience. I agree with Roberts that antigermicidal solutions and packing are without demonstrable influence, and that the packing method of dressing simple wounds in the mastoid, while more certain of thorough and solid healing, prolongs the dressing period and tends toward increasing the deformity. Were it not that delayed healing occurred under both methods of after-treatment, I should be inclined to feel that our present method was possibly at fault in the production of such an unfortunate result as a postauricular fistula or a protracted delay in the healing, which Graham-Brown classified as one of the failures of the operation on the mastoid. But such is not the case, as delayed healing was as much in evidence following the so-called "open" method.

REPORT OF CASES

A brief detail of five cases may be of interest. The first was my earliest, and as I have been unable so far to locate the case history, the recital must be entirely from memory.

CASE 1.—The simple operation on the mastoid was followed by a rather stormy course, but ultimately a normal temperature and the discharge of the patient, a child of 10, from the hospital in ten days. The discharge from the ear and that from the wound in the mastoid persisted. There was almost a complete loss of the tympanic membrane and a marked impairment of the hearing. A radical operation was performed on the mastoid at the end of six months, and a favorable result was secured, although there was, of course, no improvement in the hearing.

CASE 2.—A school teacher, aged 34, underwent a simple operation on the mastoid. The mastoid was large and pneumatic, and was filled with pus and some granulation tissue. There was little or no breaking down of the structure of the cells. The wound was dressed open. The temperature was normal five days following the operation. The patient was discharged from the hospital on the twelfth day. The ear was dry on the twenty-first day, but the perforation was still open. The wound was apparently healing nicely. At the end of three months, after stimulation of its edges with trichloracetic acid, the perforation in the anterior inferior quadrant of the membrane had finally closed, but the wound in the mastoid was still open and bare bone could be felt in the posterior part of the mastoid cavity. There was little or no discharge from the wound, and in spite of packing and cauterization of the margins of the wound, the edges continued to grow down into the mastoid cavity. Although the cavity was very large, the edges of the skin after growing down part way started to bridge across the cavity and ultimately closed it, the scar tissue stretching across the underlying space like a drum head. Thus, the posterior wound was healed three and one-half months after operation. The patient complained only of occasional tinnitus. The wound has remained in this condition since, twenty years after the operation, and has caused no trouble or inconvenience.

CASE 3.—A returned soldier, aged 21, underwent a simple operation on the mastoid following measles. The mastoid was large and pneumatic. The entire mastoid was filled with pus and granulation tissue. The dura and sinus were bare; the latter was covered with granulations. The wound was packed open. There was an apparently uneventful recovery. The ear was dry on the eighth day. The patient was discharged from the hospital on the tenth day; there was still some discharge from the wound in the mastoid. Healing was extremely slow. The discharge persisted for four months, though the ear was dry. There was an occasional slight rise in temperature. At this time, as bare bone was still felt, the patient was again referred to the hospital, where the mastoid cavity was thoroughly curetted. The wound was packed open. The patient was discharged from the hospital five days later; the ear was still dry. Six months after the original operation, the ear was dry, and there was a slight discharge from the wound in the mastoid and a small sinus leading down to the antrum of the mastoid. The sinus did not heal until eight months after the original operation. The wound had since remained closed, with no subsequent discharge—fourteen years following operation. There was only a slight impairment of hearing. There was a markedly depressed scar.

CASE 4.—A physician, aged 55, underwent a simple operation on the mastoid. The mastoid was large and pneumatic, and contained much pus under extreme pressure. There was very little granulation tissue or breaking down of the structure of the cell, with the exception of a perforation through the posterior aspect of the tip into the substance of the sternomastoid muscle. The wound was closed, except for a space for the insertion of a cigaret drain. The usual postoperative treatment was administered, with discharge from the hospital on the ninth day; there was still a discharge from the middle ear and the wound. Three and one-half weeks after the operation, the ear was dry, though there was still some discharge from the wound in the mastoid; then suddenly the patient complained of severe pain and restlessness and showed a slight fever. He was accordingly referred to the hospital, where roentgen examination was made. As this examination showed cells extending into the zygoma, these cells were opened under gas anesthesia, the sinus and dura were uncovered, and the mastoid was thoroughly explored for possible unopened cells. None were found, nor was any pus found in the cells of the zygoma. The patient was discharged from the hospital five days after the second operation. There was still some discharge from the ear and from the wound in the mastoid. The discharge persisted; there was no fever, but there were occasional attacks of severe pain. The sinuses were normal. Three weeks after the patient had left the hospital, there was no fever, but there was an occasional severe pain and a well marked diplopia on looking to the left, the side on which the operation had been performed; in other words, there was a typical Gradenigo symptom complex. This was evidently the cause of the pain when the second operation had been performed on the mastoid, though the diplopia had not developed at that time.

Two months after the operation, there was still a slight discharge from the wound in the ear and in the mastoid, and the diplopia, though decreased, was still present so that the patient could not drive his automobile. Three months after the original operation, the ear was dry, the perforation closed and the wound in the mastoid covered with a thin, tense scar similar to that described in case 2. The diplopia gradually disappeared. The hearing was normal.

CASE 5.—A housewife, aged 25, was six months pregnant. She underwent a simple operation on the mastoid. The mastoid was of fair size and pneumatic, and contained much pus and granulation tissue. No difficulty was encountered at the time of operation, except for rather profuse and persistent bleeding from the tip of the mastoid cavity, the sinus lying well forward. Clips were applied to the skin, and a cigaret drain inserted. The patient was discharged from the hospital on the sixth day. The ear was dry, and the perforation had healed; there was some discharge from the wound in the mastoid. Six weeks later, the wound was almost healed and the ear was still dry, though the patient complained of almost constant headache. Examination of the eyes showed some astigmatism. The sinuses were normal.

Two and one-half months after the first operation, following an increase in the discharge from the wound in the mastoid and considerable redness, the patient was again referred to the hospital, where the wound was reopened. The mastoid was thoroughly curetted; no dead bone was found. Clips were applied to the skin, and a cigaret drain inserted. The patient was discharged from the hospital four days later. The discharge from the wound in the mastoid persisted. The baby was born at full term. At that time the ear was dry. A conversational voice could be heard at a distance of 6 meters on the right, and at 4 meters on the left. The tympanic membrane was about normal.

Four months later, the wound in the mastoid, on account of a marked redness, was again opened with the patient under procaine hydrochloride anesthesia. Three months later it was again opened under gas anesthesia, and the dura and sinus were uncovered; the wound was left open and packed with iodoform gauze. During this period, a probe could be freely introduced into the antrum of the mastoid. If the packing was left out, the patient immediately complained of pain, headache, nausea and vomiting.

On February 28, one year and one month after the original operation, the mastoid was again opened under gas anesthesia. The patient at this time complained of headache, nausea, vomiting and severe pain behind the left eye, with inability to read, but no diplopia. At this time the sinus and dura were uncovered more freely, but nothing abnormal was found. A few small cells were found lying posterior to the sinus and a considerable number of cells above the external canal and extending well forward into the zygoma. A portion of the posterior wall of the canal was also removed. The wound was packed open. The discharge continued, the wound healing slowly. The patient otherwise was in excellent health.

At this time, following the suggestion of Fenton, the wound was packed with dichloramine-T in chlorinated paraffin. An attack of tonsillitis was followed by an increase in the discharge, but this subsequently decreased. Six months later, though the packing had been discontinued, there was still a slight mucous discharge from the wound and a small sinus leading down to the antrum. Five months later, or one year and nine months following the original operation, the ear was still dry and the wound in the mastoid had healed. Hearing was normal.

COMMENT AND CONCLUSIONS

1. In a certain proportion of cases of simple operation on the mastoid in acute mastoiditis, healing, owing to causes apparently beyond knowledge and control, will be slow and protracted.

2. In such cases, when the middle ear remains dry, one should turn to the radical operation on the mastoid only as a last resort, on account of the resulting serious impairment of the hearing.

3. In such cases, severe pain may be an indication of the Gradenigo complication, a localized inflammation at the tip of the petrous portion of the temporal bone with involvement of the gasserian ganglion. This would certainly seem to have been the explanation in one of my cases, and is a probable explanation in the last, although the third symptom in the Gradenigo triad, the diplopia, did not develop.

4. In every case of acute mastoiditis all cells should be thoroughly exenterated and subsequently secondary operations resorted to only when urgent symptoms supervene.

5. The ultimate restoration of hearing is a consideration that should always be kept in view. If there is no discharge from the middle ear and the aditus is kept open, the prognosis for the hearing is good.

6. The method of treatment described, as long as sound surgical principles are followed, apparently has no effect in the causation of delayed healing.

ADENOIDECTOMY UNDER LOCAL ANESTHESIA*

ROY F. NELSON, M.D.
OAKLAND, CALIF.

That hypertrophied and infected adenoids (pharyngeal tonsils) may be of importance in adults has been noted.[1]

Local anesthesia for tonsillectomy is being more widely used, and that its advantages should be sacrificed when performing adenoidectomies seems wrong. Nevertheless, adenoidectomy under topical anesthesia alone is very painful; it is a shock to any but hardy nerves, and is apt to be incomplete because the surgeon does not wish to punish his patient with the repeated attempts and digital exploration usually required for a thorough operation. I speak from personal experience in three painful operations of this kind, in the first two of which the results were unsatisfactory.

The following technic gives as complete anesthesia for adenoidectomy as has been obtained in tonsillectomy.

METHOD

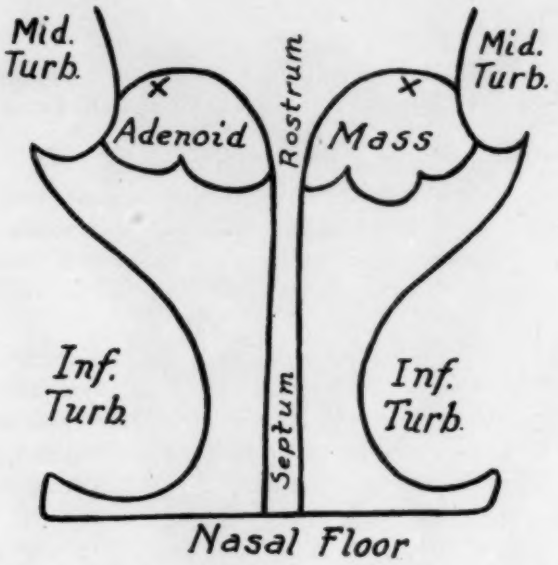
The pharynx is anesthetized as for tonsillectomy. With a curved metal applicator, the nasopharynx is swabbed transorally with cocaine in the strength preferred. The inferior turbinate is shrunk and lightly anesthetized, and the choanal vault and lateral nasal wall behind the middle turbinate and below the sphenopalatine ganglion is well anesthetized by topical applications.

With good light, the nasopharynx can now be seen through each choana, and the adenoid mass can be seen hanging below its vault. (Incidentally, in children this visibility of the adenoid after nasal shrinkage is often a great aid, especially in examining for recurrent adenoid, when the gag reflex makes posterior rhinoscopy impossible.) With a 22 gage, 4 inch (10.16 cm.), straight Luer's needle and a 2 cc. Luer's syringe, 1 or 2 cc. of 1 per cent procaine hydrochloride can now be injected beneath the adenoid on each side, inserting the needle just behind the upper rim of the choana (see accompanying illustration), and advancing the point about 1.5 cm. as the solution is slowly injected. In five minutes adenoidectomy

* Submitted for publication, Nov. 28, 1930.

1. Nelson, Roy F.: Adenoids in Adults, Arch. Otolaryng. **10**:70 (July) 1929.

can be performed and digital exploration done as thoroughly and as painlessly as under general anesthesia. A postnasal pack or string sponge may be applied for ten minutes if desired.



Drawing of the field of operation, showing the places (marked by x) for the local injection of anesthesia.

This technic is not feasible with a low deviation of the septum, unless resection of the submucous membrane is first performed, but adenoidectomy can easily accompany the latter operation. Adenoid masses confined to the fossae of Rosenmüller also are often not accessible.

411 Thirtieth Street.

METHOD OF OUTLINING MUCOCELE BY THE INJECTION OF IODIZED OIL*

G. EDWARD TREMBLE, M.D.
Associate, Department of Otolaryngology, Royal Victoria Hospital; Assistant Demonstrator, Department of Otolaryngology, McGill University
MONTREAL, CANADA

Until a few years ago mucocele of the paranasal sinuses was considered an uncommon occurrence; but owing to increasing knowledge and improved methods of diagnosis the condition is becoming more recognized. Cases are reported more frequently in the recent literature than in previous years.

Formerly symptoms of swelling in the orbital region were often diagnosed as cysts of the bone, which on closer examination at a later date proved to be mucocele. Howarth,[1] in the Hunterian lecture before the Royal College of Surgeons of England, gave credit to Logan Turner of Edinburgh and Gerber of Germany for "elucidating the condition and placing it upon a scientific basis."

Mucocele is an accumulation and retention of a mucous secretion within a sinus associated with obstruction of its outlet. According to Turner,[2] a previous chronic nasal catarrh is usually the reason for the inflammatory changes that take place, causing obstruction of the ostium of the sinus. The mucus then continues to secrete and accumulate, resulting in a thinning and absorption of one or more of the bony walls. Direct trauma and occlusion of the ostium due to an osteoma are also given as causes. The etiology varies; however, the essential feature is that the condition is due to more or less complete blockage of the ostium.

Theoretically, any of the sinuses may be affected, but in the majority of cases the frontal or anterior ethmoidal cells are involved. Cases have been reported in which the mucoceles arise from the maxillary antrum, but it is now thought that most of these were cysts of dental origin which gradually obliterated the antrum.

Mucocele of the sphenoidal sinus is such a rarity that many observers doubt its existence.

---

* Submitted for publication, Jan. 21, 1931.
* Read before the Montreal Medico-Chirurgical Society, Nov. 21, 1930.

1. Howarth, W. G.: Mucocele and Pyocele of the Nasal Accessory Sinuses, Lancet **2**:744, 1921.

2. Turner, A. Logan: Mucocele of the Accessory Nasal Sinuses, Edinburgh M. J. **22**:396 and 481, 1907.

CLINICAL FEATURES AND SYMPTOMS

The patient usually notices a painless swelling which appears at the inner and upper angles of the orbit. The sensation elicited varies from bony hardness to a parchment-like feeling or fluctuation, depending on the amount of absorption of bone. A true mucocele is not tender on gentle palpation, and there is an absence of pain or redness unless it becomes infected (pyocele).

The skin overlying the swelling remains unchanged and is freely movable. Pressure does not reduce its size. The slow progress of the swelling is characteristic until the bone becomes eroded, when it usually develops rapidly. Symptoms of pressure on the orbital contents appear, and, when present, the eyeball is displaced downward and outward. Diplopia is sometimes present, but it is not a constant feature and it is occasionally surprising to see a great degree of displacement of the globe without double vision. When it does occur, the images are seen one above the other. The ophthalmologic examination usually gives negative results; movements of the eyeball are not, as a rule, affected, the pupil reacts to light, and the fundus is normal. When pressure from the swelling involves the nasolacrimal duct, epiphora is an early symptom, and this together with the orbital displacement frequently causes the patient to consult an ophthalmologist. A lack of definite symptoms is characteristic of this condition. Headache is perhaps the chief complaint although, as a rule, it is not localized. More often the patient complains of a generalized ache. Exophthalmos is occasionally present, depending on the situation of the swelling and the amount of pressure on the orbital contents.

These cases are not sufficiently rare to report unless there is something unusual in their character. The fact that iodized poppyseed oil 40 per cent was used to outline the extent of the mucocele and the result obtained prompted the reporting of the following case. In the literature, no mention was found of the use of iodized oil, although several hundred cases have been reported. However, as the use of opaque oil has come more into prominence in rhinology during the past few years, this is not surprising.

REPORT OF CASE

A man, aged 29, complained of an occasional right frontal headache, a slight swelling below the right eyebrow of three months' duration and that the right eye was "not quite straight."

There were no other symptoms; that is, there was no impairment of sight or double vision, no previous nasal discharge and no history of trauma.

Examination showed a moderately firm, circumscribed swelling, about the size of a small grape, at the inner and upper angle of the right orbit. There was no redness, the skin over the swelling being freely movable and unaltered. All movements of the globe appeared normal, although the right inner canthus was

0.3 cm. lower than the left. Gentle palpation of the swelling did not alter its size or cause pain, although there was slight tenderness on firm pressure.

Intranasal examination showed nothing abnormal. On transillumination, the right frontal sinus appeared darker than the left. Vision in both eyes was perfect, and the fundi were normal.

The patient was advised to have the usual external operation; the alternative intranasal method of breaking down the anterior ethmoid cells and establishing free drainage was also explained. The latter procedure strongly appealed to the patient, as he was anxious to return to work as soon as possible. The anterior end of the right middle turbinate was removed under local anesthesia. On removal

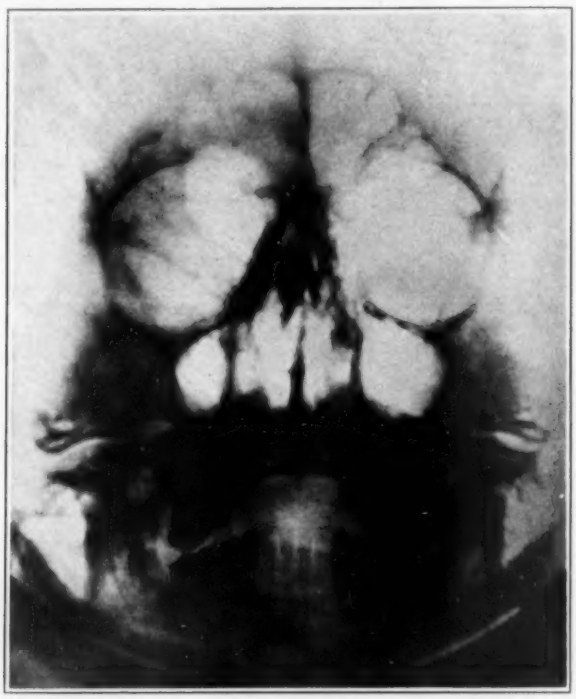


Fig. 1.—Anteroposterior view, showing the mucocele encroaching on the right orbit.

of the bone, a few drops of glairy, chocolate-colored fluid appeared near the attachment of the middle turbinate with the lateral wall. On gentle manipulation, by means of a cotton-tipped applicator, the opening was found and the depth of the mucocele measured. The probe extended inward 9 cm. from the nasal spine, practically straight backward and upward at an angle of 45 degrees. A flexible cannula attached to a suction machine was introduced and the fluid in the mucocele aspirated. This was done in order to obtain a little of the contents for bacteriologic examination, as most of the cases reported showed sterile fluid on culture. The result proved to be the same in this case. Aspiration of the cavity suggested the possibility of injecting iodized oil through the cannula in order to

Fig. 2.—Lateral roentgenographic view. The mucocele can be seen extending backward into sphenoidal sinus.

Fig. 3.—Roentgenogram taken after the injection of iodized oil.

outline the whole extent of the mucocele. This was accomplished with the patient lying on the table, the chin being raised to an angle of about 15 degrees. As the caliber of the cannula was considerably less than the opening into the mucocele, there was sufficient space for the escape of air. After inserting the cannula, the oil was injected slowly, exactly in the same manner as is used in outlining the frontal sinus. On the appearance of the overflow in the nose, the cannula was removed and a small moistened pack of absorbent cotton placed in the opening to prevent the oil from leaking out. Roentgenograms both anteroposterior and

Fig. 4.—Side view, showing the mucocele filled with iodized oil and the location of the ostium.

lateral views, were taken immediately, with the results shown in the accompanying figures.

Although the anteroposterior plate shows that the mucocele extended well above the roof of the orbit, no connection could be found with the frontal sinus. The lateral view, especially figure 4, shows that the mucocele was ethmoidal; the majority of recorded cases appear to be of this type (usually the anterior group). Superiorly, the wall of the cavity blended with the floor of the anterior and middle fossa, while posteriorly it extended through the sphenoidal sinus and approached the pituitary fossa.

COMMENT

Although the injection of iodized oil proved successful in this case, one would hesitate to use it without previous x-ray plates, in order to be certain that the mucocele had not eroded into the anterior or the middle fossa.

In conclusion, it might be mentioned that in this case only the simple intranasal opening was made. The chance of recurrence was explained to the patient, but he promised to return and to undergo the external operation if there was any further need.

Under local anesthesia, the ostium of the mucocele in the middle meatus was enlarged and the anterior ethmoid cells curretted away. A large drainage tube was inserted and held in place for ten days. Nothing further was done. The patient was kept under observation to note the size of the swelling in the orbit from time to time.

Within a month, the eye had returned to its normal position, and the swelling had disappeared.

1390 Sherbrooke Street.

ANAPHYLACTIC CHANGES IN THE NASAL MUCOSA OF GUINEA-PIGS *

WILLIAM E. MURPHY, M.D.
BOSTON

My purpose in this paper is to record experiments on animals undertaken to determine what constant changes, if any, occur in the nasal mucous membrane in anaphylactic shock. The work was carried out at the Otolaryngological Laboratory of the Massachusetts Eye and Ear Infirmary.

It is well known that animals can be sensitized to foreign proteins. Guinea-pigs were selected for these experiments because of the ease with which they may be rendered sensitive. Ten animals in all were used, and eight were sensitized to two different allergens, two animals being reserved for controls. Five were given intraperitoneal injections of 0.2 cc. of horse serum, and the other three, a very dilute solution of filtered egg albumin, the nitrogen content of which was not determined. After the injections were given, an interval of two weeks in the case of those given horse serum and of three weeks in those given egg albumin was allowed to elapse so that complete sensitization might take place. At the end of this period, the animals were again given intraperitoneal injections of 5 cc. of the respective sensibilisinogens employed to produce the allergic condition. An anaphylactic reaction was elicited in six of the eight animals, and no apparent changes were manifested in the other two. The severity of the anaphylactic phenomenon was well demonstrated by changes which were produced with egg albumin. Starting within a few minutes after the injection, gross symptoms occurred rapidly, reaching a climax within twenty minutes. One of the animals failed to react. The animals given horse serum reacted in a lesser degree, and death did not take place for several hours. After a moderately severe paroxysm lasting for two hours one guinea-pig recovered, but was killed twenty-four hours later with potassium cyanide. Another failed to show any response.

SYMPTOMATOLOGY

The gross symptoms were characteristic in all cases; the severity was commensurate with the violence of the reaction. First there was a preliminary or incubation period lasting from one to several minutes. This

* Submitted for publication, Jan. 5, 1931.

* From the Otolaryngological Laboratory of the Massachusetts Eye and Ear Infirmary.

was followed by itching of the nose and at the site of the injection, as shown by the violent scratching of these areas. The respiration became accelerated; the fur appeared ruffled and stood on end, and urine and feces were discharged. Marked weakness set in; the hind legs were affected first. The breathing became labored, irregular, shallow and slow. The animal fell to one side, and the muscles of the legs and trunk twitched irregularly. Deep cyanosis developed. This was most noticeable on the extremities and about the nose and mouth. Convulsions set in, and the animal often attempted to rise and run, heedless of any sense of direction. Death usually followed after one or

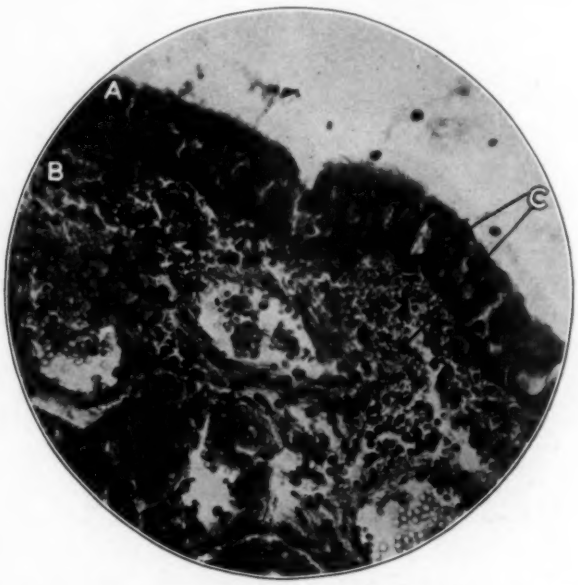


Fig. 1.—The nasal mucous membrane of a guinea-pig that died from anaphylactic shock three hours after the injection of horse serum. *A* indicates surface epithelium; *B*, blood vessels, and *C*, petechial hemorrhage in the propria just

York, The Macmillan Company, 1914, chapters on anaphylaxis.

two of these paroxysms. At autopsy the well known observation, namely, distended, pale bloodless lungs which filled the thorax, was present. Sections of the lungs showed heaping up of the mucous membrane and numerous small hemorrhages around the bronchioles.

MICROSCOPIC OBSERVATIONS

The mucous membrane from the nose and the adjacent structures was removed, fixed and stained by the ordinary procedures for examination. The normal mucous membrane of the guinea-pig resembles that of man. The epithelium is ciliated and pseudostratified and lies on a

thin band of basement membrane. There is a well developed tunica propria which consists of fibrillar and fine elastic tissue. This is especially abundant in the deeper layers and contains many mixed, branched, tubulo-alveolar glands and a few lymphocytes that occasionally form a solitary node. The tunica propria is well supplied with blood vessels and forms a dense network or plexus subepithelially.

On microscopic examination, certain changes were marked. In the animals in which the symptoms of anaphylaxis lasted several hours, the changes were well defined. The following microscopic observations were made: Certain areas of the epithelial tissue showed distended

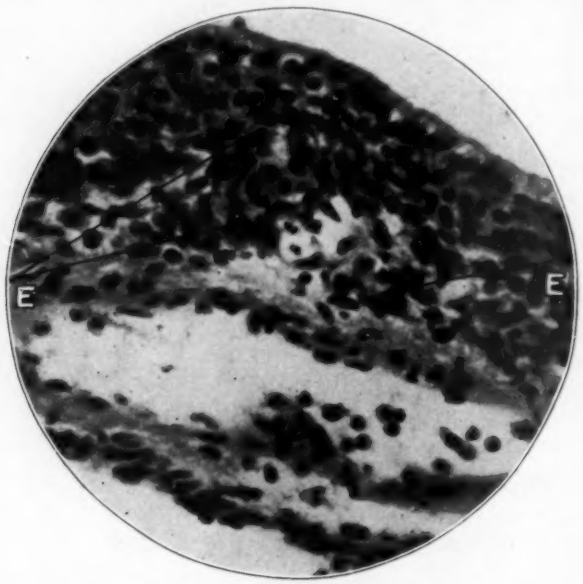


Fig. 2.—The nasal mucous membrane of a guinea-pig killed with potassium cyanide twenty-four hours after recovery from anaphylactic shock produced by the injection of horse serum. *E* indicates dense eosinophilic infiltration in the subepithelial tissue.

goblet cells, while other goblet cells were discharging their contents. More striking changes were found beneath the membrana propria in the subepithelial tissue. Sections from the guinea-pig that was killed with cyanide showed a dense infiltration of eosinophils. Scattered infiltrations of eosinophils were found in the other sections. All of the blood vessels beneath the membrana propria were dilated and filled with blood. There was a generalized hyperemia in the plexus of blood vessels. The chief and most important observation beneath the basement membrane, however, was the presence of numerous petechial hemorrhages.

COMMENT

The animals that were sensitized with a powerful allergen reacted more quickly and to a severer degree than those in which weaker allergens were used; hence, the anaphylactic process developed to a maximum point, with death ensuing in a few minutes. In these cases due to the rapid transition from the initial to the final stage, there was relatively little chance for many changes to take place; therefore, the pathologic changes were in direct proportion to the reaction elicited. The gross symptoms may be attributed to the influence of the stimulation of smooth muscle. It has been shown by a number of workers experimenting on various animals that such stimulation is a prime factor in the phenomenon of anaphylaxis. It is believed that the action is on the muscle cells themselves rather than on the nerve endings. The work of Biedl and Kraus [1] tends to confirm this view.

The etiology of the petechial hemorrhages is somewhat obscure. Several factors may be involved. Certain endothelial changes plus spasm of the walls of the arterioles could account for this pathologic change. In their studies, Gay and Southard [2] referred to local fatty changes in the capillary endothelium that may play a part. No plausible explanation is offered for the animals that failed to react, other than that they failed to become sensitized.

CONCLUSIONS

Anaphylaxis in the guinea-pig is manifested by definite and characteristic symptoms. They may be severe and of short duration or mild and drawn out. Death does not necessarily follow an attack. The microscopic observations in the nasal mucous membrane are pronounced. They are as follows: eosinophilia, congestion and, especially, petechial hemorrhages.

Dr. T. B. Mallory gave me suggestions for this paper and reviewed the slides.

---

1. Biedl and Kraus, in Zinsser, Hans: Infection and Resistance, ed. 3, New York, The Macmillan Company, 1914, chapters on Anaphylaxis.

2. Gay and Southard: On the Method of Serum Anaphylaxis and Intoxication in the Guinea Pig, J. M. Research **13**:410, 1908; On Serum Anaphylaxis in the Guinea Pig, ibid. **11**:161, 1907.

RETROBULBAR OPTIC NEURITIS ORIGINATING IN THE NASAL SINUSES

A NEW METHOD OF DEMONSTRATING THE RELATION BETWEEN THE SPHENOID SINUS AND THE OPTIC NERVE *

HARRIS H. VAIL, M.D.
CINCINNATI

In spite of the fact that ophthalmology and rhinology seem to have become two separate and distinct specialties, they have so many interesting and difficult problems in common that there will always be a need for close and intelligent cooperation between those practicing these two branches of medicine.

This need is real in the problem of retrobulbar optic neuritis originating in the nasal sinuses. This condition has been studied and speculated on for hundreds of years. According to Canuyt, Ramadier and Velter,[1] Baratoux [2] stated that in the eleventh century, Ali-Ibu-Isa, a Persian, described the engorgement, compression and inflammatory swelling of the optic nerve resulting from a nasal "catarrh"; in the eighteenth century and first two thirds of the nineteenth, there appeared from time to time, reports of cases which showed that there was an understanding of the relation between certain ocular symptoms and disease of the nose and sinuses. In all these observations, however, the concomitance of ocular disturbances and suppuration of the sphenoid and ethmoid sinuses was found only at autopsy. By 1877, clinicians began to look for a relation between certain obscure visual troubles and disease of the posterior sinuses, and in 1886, Berger demonstrated perineuritis of the intracanalicular portion of the optic nerve of sphenoethmoid origin.

Canuyt, Ramadier and Velter quoted Ziem as writing in 1892, "In ten years, one will not be able to practice ophthalmology without rhinology," and "two thirds of the ocular affections have their origin in an unrecognized rhinopathy." They mentioned the work of Grünwald in 1893, the article of Parinaud on retrobulbar neuritis in 1896, and the impetus given to the subject by Delapersonne who stated, in

* Submitted for publication, Oct. 10, 1930.

* Read at a meeting of the Chicago Laryngological and Otological Society, Dec. 1, 1930.

1. Canuyt, Ramadier and Velter: Les sinusitis postérieures et leurs complications oculaires, Ann. d. mal. de l'oreille, du larynx **44**:39 (Jan.) and 140 (Feb.) 1925.

2. Baratoux: Monographies médicales, Dec. 15, 1923.

1898, that one should suspect and examine the sphenoid sinus in cases of ocular symptoms of undetermined origin, and who reported three cases of optic neuritis resulting from spheno-ethmoiditis without any of the usual signs of sinusitis.

Since then, the literature of retrobulbar optic neuritis has become quite extensive, the names of Sluder, Leon White, Loeb and Jonathan Wright standing out among the American writers.

To White [3] great credit must be given. He consistently worked on the subject, and published many important contributions the first of which appeared in 1916.

However, the mechanism of the production of retrobulbar optic neuritis of nasal origin has not been proved clinically or experimentally, and therefore has become a much discussed question. The theories which have been advanced are: (1) a toxic action on the papillo-macular bundle; (2) direct extension of the infecting agents from the posterior sinuses to the optic nerve through dehiscences or along blood and lymphatic vessels; (3) hyperplastic bony changes with resulting pressure on the optic nerve in the optic canal.

None of these seems to explain satisfactorily the clinical picture. One important factor has been the absence of suitable material for autopsy. Practically all the microscopic studies were done on tissue removed at the time of operation, mostly bits of the anterior wall of the sphenoid sinus.

In 1928, Prof. H. Herzog,[4] of Innsbruck, Austria, demonstrated, I believe, the real mechanism of the production of retrobulbar optic neuritis originating in the nasal sinuses. He examined 435 chronic and 65 acute cases of disease of the nasal sinuses between the years 1919 and 1927. Thirteen cases, or 2.6 per cent, presented definite characteristics of retrobulbar neuritis. Of these 13 cases, 6 occurred in chronic inflammation of the sinuses, and 7 in acute inflammation. In other words, according to Herzog, changes of the optic nerves were present in acute sinusitis from 7 to 8 times more frequently than they were in chronic sinusitis.

Herzog did not have any cases of retrobulbar optic neuritis that came to autopsy, but in sections of other cases he found that when there were extensive marrow spaces of the bone between the optic canal and the sphenoid sinus, these were intimately connected with the submucosa of the sinus and the dura of the sheath of the optic

3. White, Leon: Loss of Sight from Retrobulbar Neuritis, Due to Accessory Sinus Disease, with a Report of Two Cases, Boston M. & S. J. **174**:790 (June 1) 1916.

4. Herzog, H.: Ueber die Neuritis retrobulbaris, Arch. f. Augenh. **99**:291 (July) 1928.

nerve by means of cellular processes running from the dura into the spaces where they blended with cellular elements derived from the submucosa of the sphenoid sinus extending into the same spaces. He found that there were many more openings into the marrow spaces from the side of the nasal sinus than from that of the optic nerve. He demonstrated a normal variation in the amount and size of the marrow spaces in the bony wall between the optic nerve and sphenoid sinus.

He showed that infection in the sphenoid sinus had extended readily into the marrow spaces from which it could easily pass along the cellular processes into the sheath of the optic nerve. He found edema with other inflammatory changes in the marrow spaces without

Fig. 1.—The Vail-Lange position for roentgenogram of the right sphenoid sinus.

any changes in the sinus mucosa. He stated that slight inflammatory changes in the superficial layer of the sinus mucosa could disappear while a severe infection continued in the marrow spaces. This was considered the explanation of the practically negative indications in the sinus.

These conditions were well illustrated by Herzog. He felt that the pathologic process in retrobulbar neuritis was one of circumscribed serous meningitis of the sheath of the optic nerve, with or without interstitial changes in the nerve.

His work was verified in one case reported by ten Doesschate.[5] The latter reported a case of chronic pansinusitis with death from

5. ten Doesschate, G.: Klin. Monatsbl. f. Augenh. **80**:831 (June) 1928.

pneumonia. Microscopic study showed purulent infiltration with diplococci in the mucosa of the sphenoid sinus, in the bone marrow of the wall between the sphenoid sinus and the optic nerve, in the dural sheath of the optic nerve and in its septums.

However, there are some who feel that retrobulbar optic neuritis originating in the nasal sinus is a rare condition; they believe that the cases are not surgical; that retrobulbar optic neuritis is usually the first symptom of multiple sclerosis; that for this reason, operations on the sinuses are not indicated, and that any restoration of vision following such surgical procedures are coincidental and merely due to the fact that there are natural periods of remission in the course of multiple sclerosis.

Scheerer [6] felt that only 1.5 per cent of the 203 cases of retrobulbar optic neuritis seen by him from 1921 to 1928 were due to disease of the nasal sinuses. He claimed that multiple sclerosis is the chief cause of retrobulbar optic neuritis. In all but one of Scheerer's acute cases the patient recovered vision without nasal operation; he felt that the prognosis for the return of vision under local and medical treatment in the acute cases was good.

Marx [7] stated that the diagnosis of retrobulbar optic neuritis originating in the nasal sinuses is often difficult to make. Often it is made, as Bruckner said, from "Wahrscheinlichkeit (probability)." To prove that the cause is in the posterior sinuses is difficult, but as a rule it is possible after careful examination. In some cases an exploratory diagnostic operation is indicated. In the differential diagnosis, multiple sclerosis is of first consideration. The space of time from the onset of the retrobulbar optic neuritis until the appearance of symptoms of multiple sclerosis may be ten years or longer.

In a group of cases in which a nasal operation was performed, improvement in vision resulted, but later multiple sclerosis appeared. However, no statistics on this subject were given by Marx.

Marx stated that Meller and Wertheim believed that in cases in which the rhinologic observations are negative and in which no other certain etiologic factor can be found, one should operate. Hajek, von Eiken and Schlesinger had the opposite view, while de Kleyn, Jung and Elschnig reported good results from conservative therapy. No radical operation is necessary in any of the cases, according to Marx.

Marx quoted Kostenbaum's series of thirty-five cases of retrobulbar optic neuritis originating in the nasal sinuses. Eight patients were

6. Scheerer, R.: Ueber die Ursachen der Neuritis retrobulbaris, Klin. Monatsbl. f. Augenh. **83**:164 (Aug.-Sept.) 1929.

7. Marx, Hermann: Die orbitalen Komplikationen bei Nebenhohlenentzünden, Handb. d. Hals-, Nasen- Ohrenh. **2**:977, 1926.

operated on and twenty-seven were not. Of the latter, eighteen recovered and two were considerably improved. Seven showed no change. Eight of the thirteen eyes operated on showed no improvement; the others had the same relative course as the eyes not operated on.

Weill [8] stated that from his researches the rhinologist practically never observes acute retrobulbar neuritis associated with posterior sinusitis and that the ophthalmologist practically never sees lesions of the sinuses in cases of retrobulbar optic neuritis. Of forty cases, he observed a progressive return of vision without surgical intervention in more than 85 per cent.

He stated: 1. The prognosis of acute retrobulbar optic neuritis even without surgical intervention may be considered favorable. 2. The origin of acute retrobulbar optic neuritis should be sought some-

Fig. 2 (case 1, Mrs. B.; diagnosis, retrobulbar optic neuritis on the left side; recurrent attacks).—*A*, anteroposterior roentgenogram with the head in the vertical position. Radiopaque oil has been injected into the left maxillary and sphenoid sinuses. The filling defect of the maxillary sinus and the very large size of the sphenoid sinus are well shown. *B*, lateral view, showing the filling of the left maxillary sinus and sphenoid sinus with radiopaque oil.

where else than in the posterior sinuses. 3. Acute retrobulbar neuritis is to be considered as the first, and often the only, sign of multiple sclerosis. 4. The condition does not call for an operation on the sinuses.

Obviously, he must have overlooked the clinical and pathologic studies of Herzog and the mass of evidence accumulated from American authorities.

---

8. Weill, G. A.: The Relationship Between Inflammation of the Posterior Sinuses and Disease of the Nervus Opticus, Arch. Ophth. **1**:307 (March) 1929.

According to Ziegler,[9] who studied 529 cases of multiple sclerosis at the Mayo Clinic from 1915 to 1927, defects of vision such as diplopia and central scotoma constituted the symptoms at onset in only 14.4 per cent. He recorded an insidious onset in 456 cases, or 86.2 per cent, and stated that the onset and recurrences are more frequent in the spring and early summer months, that long remissions may occur, but that complete remission of all symptoms is rare. Death occurred in 136 (25.7 per cent) of the 529 cases. The average length of life from the onset of the disease until death was over seven years, in the cases on which data could be obtained.

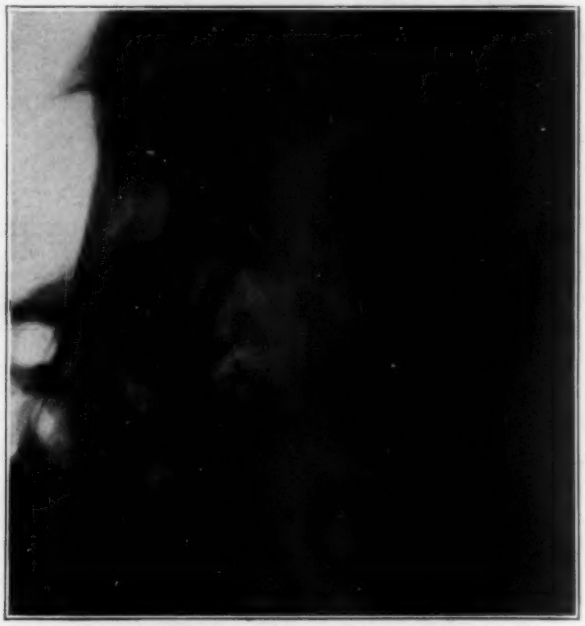


Fig. 3 (case 1).—Roentgenogram showing the relation of the sphenoid sinus to the optic canal by the Vail-Lange position. From a careful study of this film it would seem that there is a slight filling defect of the upper outer angle of the sphenoid sinus.

Ziegler's observations would seem to refute the belief that retrobulbar optic neuritis is the first and only sign of multiple sclerosis.

Is it not possible for infecting agents to gain access to the central nervous system from a site in the posterior nasal sinuses, with visual disturbance as the first and most natural symptom? If bacteria spreading from a sinus focus can involve the optic nerve, cannot the virus

9. Ziegler, L. H.: Multiple Sclerosis: A Clinical Review and Follow Up Study, Minnesota Med. **12**:778 (Dec.) 1929.

of multiple sclerosis act in the same way? Shall one deprive patients of a possible chance of avoiding or postponing the onset of multiple sclerosis by neglecting to drain surgically those nasal sinuses which are nearest the optic nerve? That multiple sclerosis may develop later is no argument against operations on the posterior nasal sinuses, and certainly the argument advanced by a few writers, that surgical intervention has been responsible for blindness and even death, and therefore should not be employed, is no argument. It should take a long

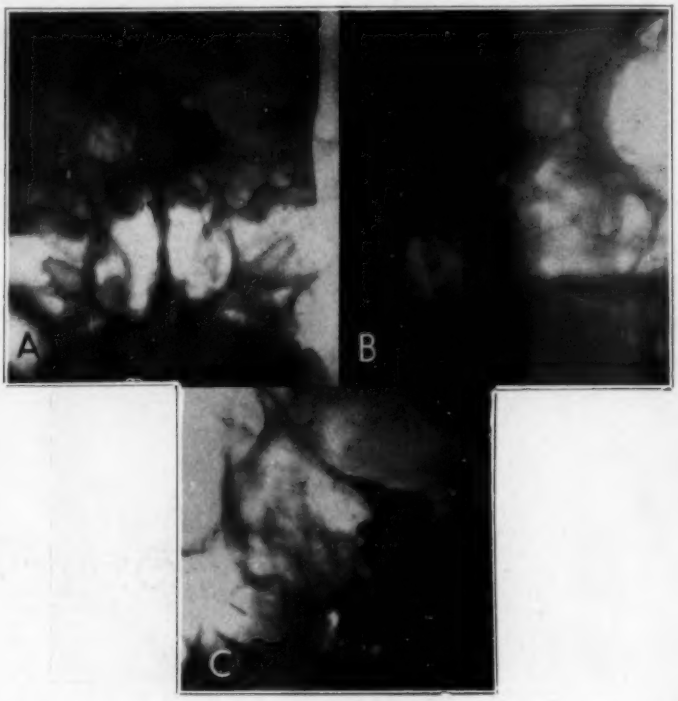


Fig. 4 (case 3, Miss D.; diagnosis, bilateral retrobulbar optic neuritis and multiple sclerosis).—*A*, radiopaque oil has been injected into the right sphenoid sinus and the films made with the head in the vertical position. Note the large size of the sphenoid sinus. *B*, a lateral view showing the large size of the right sphenoid sinus. *C*, the right sphenoid sinus has been filled with radiopaque oil and the roentgenogram made in the Vail-Lange position. Note the absence of any filling defect and the intimate relation of the radiopaque oil to the optic canal.

time for the virus of multiple sclerosis to spread throughout the central nervous system and for the other symptoms of the disease to develop. The papillomacular bundle is a very delicate nervous tissue. The various other nervous tissues affected in multiple sclerosis are more resistant.

Remissions in the course of the disease can be explained by remissions in the infection of the nasal sinuses. Similar remissions are encountered in retrobulbar optic neuritis of obvious nasal sinus etiology.

One encounters somewhat analogous conditions of the central nervous system in epidemic encephalitis (lethargica), serous meningitis, chronic arachnoiditis and various psychoses, all of which can be caused by an inflammation in the nasal accessory sinuses.

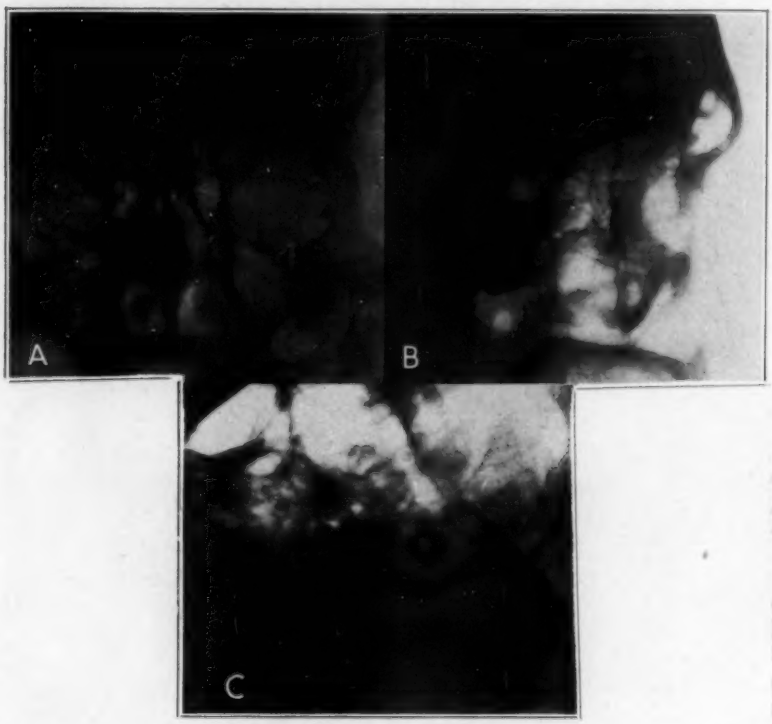


Fig. 5 (case 4, Mr. V.; diagnosis, bilateral retrobulbar optic neuritis and multiple sclerosis).—*A*, radiopaque oil has been injected into the left sphenoid sinus; note the large size of the sinus. *B*, a lateral view showing the large size of the sphenoid sinus. *C*, the Vail-Lange position. Note the absence of a filling defect and the intimate relation of the radiopaque oil to the optic canal.

In the years 1920 and 1921, I was fortunate, as an intern in the Massachusetts Eye and Ear Infirmary, in being able to work under Dr. Leon White. I saw a great many of his cases, was present at his operations and performed some under his guidance. I could not help but be inspired by the high quality of his work.

When I returned to Cincinnati in 1921, I had a further opportunity in being associated with my father, Dr. Derrick T. Vail. From his large ophthalmologic practice, I obtained the material concerning which I report.

In the years 1921 to 1929, inclusive, twenty-three cases diagnosed by Drs. D. T. Vail, Sr., and Jr., as retrobulbar optic neuritis were referred to me. Of these twenty-three, eight were found after careful

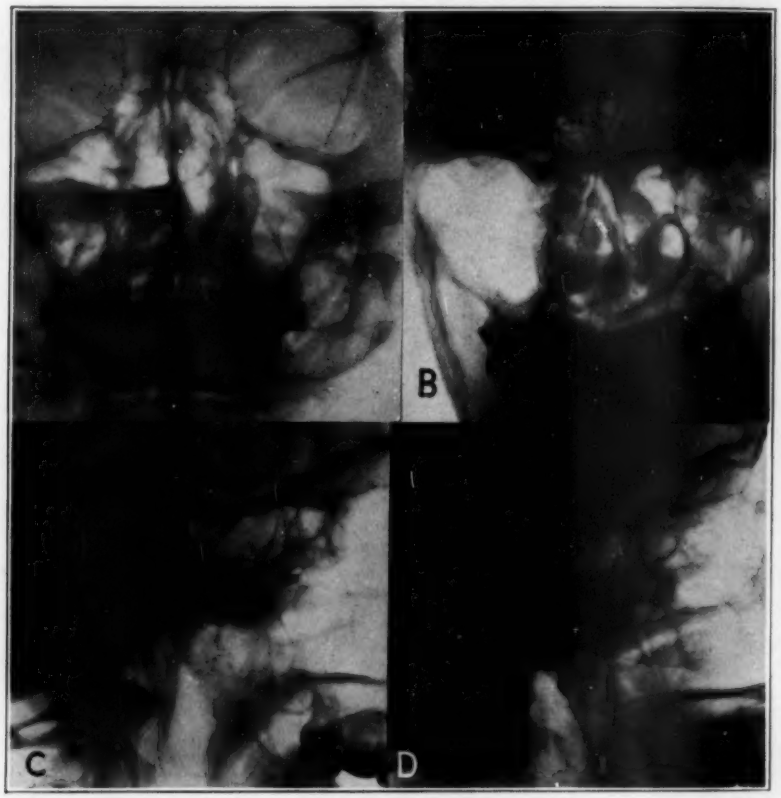


Fig. 6 (case 5, Mrs. S.; diagnosis, central choroiditis and chronic tonsillitis; disease of the sinuses not demonstrable clinically).—*A*, roentgenogram of the left sphenoid sinus after the injection of bromipin 33 per cent; the head is in the vertical position. Note the large size of the sphenoid sinus. *B*, anteroposterior roentgenogram made after the injection of iodized poppy seed oil 40 per cent in the right sphenoid sinus, with the head in the vertical position. The shadow of the bromipin in the left sphenoid sinus is dim. The right sphenoid sinus received an injection of iodized oil three hours after the bromipin was injected into the left sinus. Note the asymmetry of the two sphenoid sinuses. *C*, lateral roentgenogram with the head in the vertical position; bromipin was injected into the left sphenoid sinus. The large size of the sinus can be seen. *D*, a roentgenogram of the right sphenoid sinus following the injection of iodized oil with the head in the vertical position. What might be taken for a filling defect is probably due to an overlapping of the left sphenoid sinus.

physical, neurologic and serologic examination by competent examiners to be due to syphilis, tobacco, lead, quinine, multiple sclerosis and pernicious anemia. The remaining fifteen were diagnosed by me as of nasal sinus etiology. A careful rhinologic and roentgen examination

TABLE 1.—*The Etiology of Retrobulbar Optic Neuritis in Twenty-Three Cases (1921-1929)*

	Origin in Nasal Sinus	Syphilis	Tobacco	Lead	Quinine	Pernicious Anemia and Multiple Sclerosis	Multiple Sclerosis
Cases.....	15	2	2	1	1	1	1
Per cent....	65.2	8.7	8.7	4.3	4.3	4.3	4.3

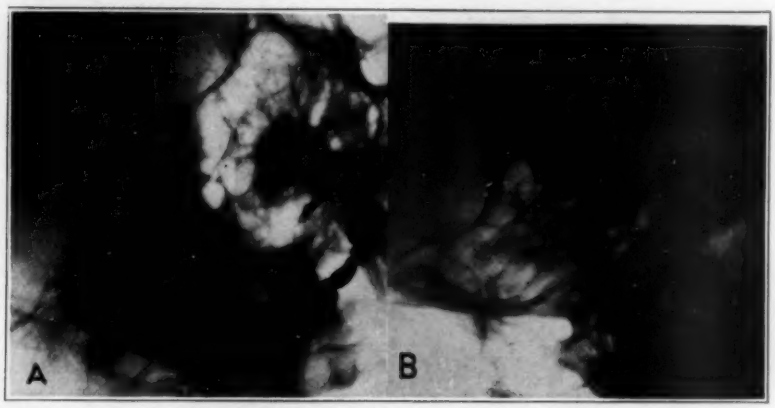


Fig. 7 (case 5).—*A*, roentgenogram of the left sphenoid sinus showing the close relation between the sphenoid sinus and the optic canal. From this film it can be definitely ascertained that the cell lying near the optic canal is not a part of the left sphenoid sinus. *B*, roentgenogram of the right sphenoid sinus in the Vail-Lange position, following the injection of iodized oil. The close relation between the sphenoid sinus and the optic canal is shown. The paranasal sinus cell near the optic canal is definitely proved not to be of either sphenoid sinus and is therefore probably a portion of the posterior ethmoid system.

was made in all cases. Negative roentgen observations were made in ten cases (66 per cent). Clouding of the ethmoids, demonstrated roentgenologically, and positive clinical indications in the nose were present in five cases (33 per cent).

According to the records of Drs. D. T. Vail, Sr., and Jr., a normal fundus was present in eight cases; optic neuritis was found in five cases and sectional optic atrophy in two of the cases of longer duration. There was a central scotoma for white and colors in the more severe

cases; in two mild cases it was for colors only. The peripheral field was normal in all cases. Visual disturbances ranged from mild central scotoma to a severe diminution of vision, but in no case amounted to total blindness.

The youngest patient was a boy aged 12 years; the oldest, a man aged 58. In three cases the retrobulbar optic neuritis was bilateral. Seven male and eight female patients were affected.

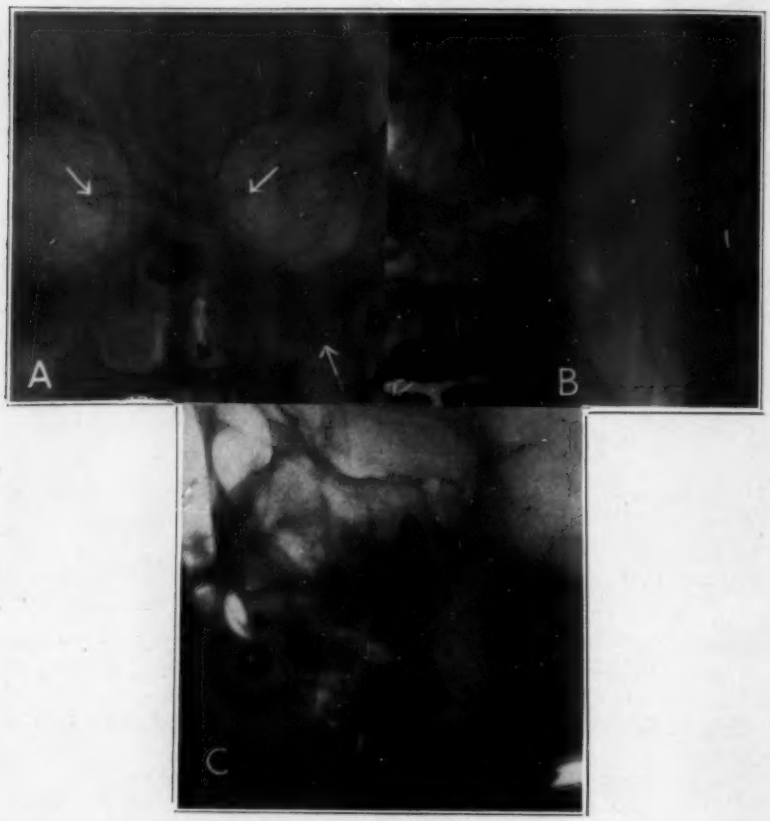


Fig. 8 (case 6, Miss C.; diagnosis, asthma and pansinusitis; polypi were found in all the sinuses at operation and transient attacks of amaurosis occurred in the right eye during severe attacks of asthma).—*A*, anteroposterior roentgenogram, following the injection of radiopaque oil in the right sphenoid sinus with the head in the vertical position. *B*, lateral roentgenogram, with the head in the vertical position. Note the filling defect of the sphenoid sinus. *C*, roentgenogram in the Vail-Lange position showing the marked filling defect of the upper outer angle of the sphenoid sinus. (The nasopharyngoscope passed into the right sphenoid sinus several months after operation demonstrated the presence of polypi, chiefly on the roof and in the upper outer angle of the sinus.)

The acuteness or chronicity of the sinusitis was determined by the history and clinical indications as follows: Four patients had no symptoms or history of sinusitis. Six patients had symptoms and a history suggesting an acute sinusitis. Five patients had symptoms and a history suggesting chronic sinusitis. One of these was a patient who, for nearly two years, had had with every cold a unilateral diminution of vision, at times severe, but with a clearing up of the blindness when the cold improved. Roentgen examination of the nasal sinuses following the injection of iodized poppy seed oil 40 per cent showed filling defects of the maxillary sinus and the sphenoid sinus on the side of the affected eye.

An interesting point was found on study of the case histories. Eleven of the fifteen cases occurred in the fall, winter and spring months, two in May and one in July. The chronic recurrent case could not be classified.

TABLE 2.—*The Relation Between the Etiology and the Extent of the Condition*

	Unilateral			Bilateral				
	Origin in Nasal Sinus	Multiple Sclerosis	Lead Poisoning	Origin in Nasal Sinus	Syphilis	Tobacco	Quinine	Pernicious Anemia
Cases.....	12	1	1	3	2	2	1	1
Per cent.....	80	100	100	20	100	100	100	100

Most interesting, I believe, of all the observations was the fact that in the cases in which the onset was acute and could be definitely dated, the blindness was noticed after a night's sleep. In five of the cases, the course was so gradual that no definite date of onset could be given by the patient. The recurrent case could not be included in this grouping.

I shall report in detail one of the most interesting of the cases.

REPORT OF A CASE

Mrs. A. A., aged 46, was first seen on March 10, 1922. She stated that one month previously, she had noticed the gradual onset and progress of defective vision in both eyes. She was not subject to colds, but about a month before, had one that lasted for three days. She had no severe headaches. Within the past eight months, she had noticed a little dizziness. Her memory had been poor for several years, but there had been no recent change. For eight months she had noticed numbness in both hands and feet. Her general health had been only fairly good.

Ophthalmoscopic examination (by Dr. D. T. Vail, Sr.) showed: On March 10, 1922, there was some swelling of both optic disks, with swelling of the retina about the disk. The macular regions were normal. In the right eye vision was 20/200, and in the left, perception of fingers at 2 feet. The visual field in each eye showed a central scotoma. The diagnosis was acute neuroretinitis (bilateral).

Urinalysis showed the smallest possible trace of albumin. The blood pressure was normal. A general physical examination (by Dr. Greiwe of Cincinnati) showed nothing definite, although the patient was not in good health. The Wassermann reaction was negative. A lumbar puncture (by Dr. Faller of Cincinnati) showed the spinal fluid under increased pressure, and globulin + 1. No cell count was recorded. Neurologic examination gave normal results.

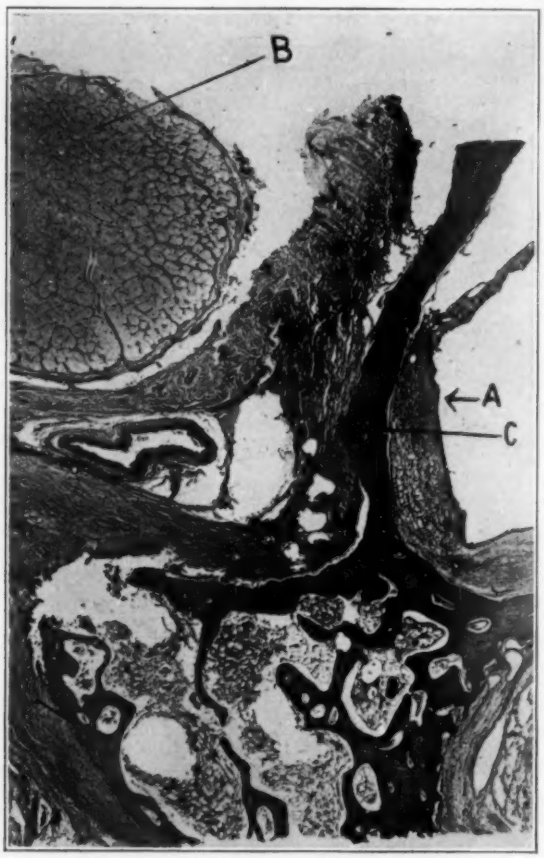


Fig. 9.—Photomicrograph showing the relations between the sphenoid sinus mucosa (*A*) and the optic nerve (*B*). This section shows a very compact bony wall (*C*) between the sphenoid sinus mucosa and the dural sheath of the optic nerve, and is analogous to figure 5 in Professor Herzog's article, Ueber die Neuritis and retrobulbaris (Arch. f. Augenh. **99**:292 [July] 1928).

There was slight deviation of the nasal septum with no obstruction to breathing. Both middle turbinates were enlarged. The nasopharynx was clear and no pus was seen in the nose. Transillumination showed both antrums bright.

A roentgen examination (by Dr. Sidney Lange of Cincinnati) showed that the frontal sinuses and antrums were clear. There was slight clouding of the right posterior ethmoid region. The sphenoid sinuses were clear.

My first impression of the case was that the retrobulbar optic neuritis did not originate in the nasal sinuses. However, when all the other possible causes seemed to be excluded, the posterior sinuses were operated on on March 17, 1922. Under ether anesthesia, a submucous resection of the septum was done. The left middle turbinate was removed, and the left posterior ethmoid cells were opened back to the sphenoid sinus. A large opening was made in the front wall of the latter. The mucosa of the sphenoid sinus was grayish. No disease was seen in the sphenoid sinus on inspection through the Holmes nasopharyngoscope passed into the cavity. The same procedure was used on the right side, and a complete absence of disease in the posterior sinuses was noted.

On the following day the patient stated that vision seemed brighter. Two days after operation, vision was 20/200 in the right eye and perception of fingers

TABLE 3.—*Observations on the Fundus, Field and Vision in Fifteen Cases Originating in Nasal Sinuses*

Fundus					Visual Disturbance		
Normal	Optic Neuritis	Sectional Atrophy	Central Scotoma	Peripheral Field	Mild	Moderate	Severe
8	5	2	15	Normal in all	5	1	9

TABLE 4.—*Seasonal Prevalence and Type of Infection*

Extreme Ages	Males	Females	Acute Sinusitis	Chronic Sinusitis	No Symptoms of Sinusitis	Seasonal Prevalence
12-58 yrs.	7	8	6	5	4	5 in March 2 in April 2 in May 1 in December 1 in January 1 in February 1 in July 1 in September 1 recurrent case

at 5 feet in the left eye; after six days fingers were perceived by the left eye at 6 feet; after eight days, at 15 feet, and after ten days, at 18 feet, vision in the right eye remaining 20/200. At this time, Dr. D. T. Vail, Sr., noted that the optic neuritis and retinitis were disappearing.

Five weeks after operation, vision was 20/200 in the right eye and 20/200 in the left; two months afterward, 20/40 in the right eye and 20/100 in the left, and seven months afterward, 20/20 in the right eye and 20/30+ in the left. Dr. D. T. Vail, Sr., noted slight optic atrophy on the temporal side of both disks.

Thirteen months after operation, vision was 20/20 in both eyes.

The patient was seen at irregular intervals during the ensuing years. Recently, almost eight years after operation, she wrote that her eyesight was normal and that, outside of a sluggish liver, she was doing well.

Comment.—A diagnosis of multiple sclerosis could not be made definitely in this case; yet it seemed that there was some disturbance of the central nervous system. The spinal fluid was under increased pressure. If operation had not been performed, would this case have

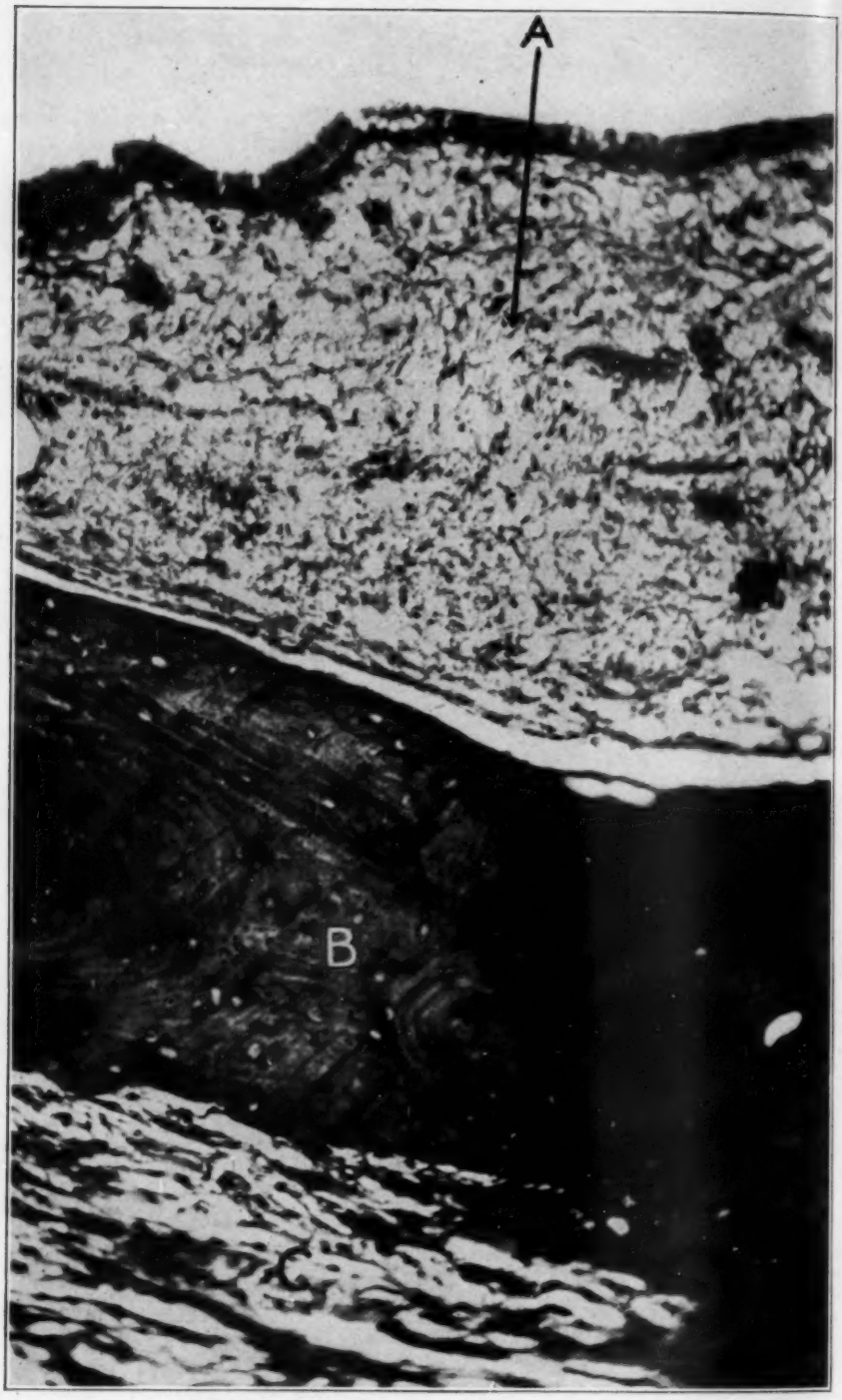


Fig. 10.—Higher magnification of the same section to show the very compact structure of the bony wall (*B*) between the sinus mucosa (*A*) and the dural sheath of the optic nerve (*C*). Figures 9 and 10 were made through the cooperation of Dr. M. Herzberg, pathologist, Jewish Hospital, Cincinnati, and Mr. J. B. Homan, assistant professor of medical art, College of Medicine, University of Cincinnati.

progressed to multiple sclerosis at a later date? Or must more years pass before the diagnosis is proved or disproved?

I did not make a roentgen examination of the optic canals until 1924, as White [10] did not publish his work on that subject until 1923. The cases in which this special roentgen examination was made showed no definite indications. Very large and average-sized canals were found in patients whose vision was severely affected. I never considered the size of the optic canal the deciding factor as to when to operate.

TABLE 5.—*Onset of Condition*

	Acute	Gradual, Undetermined	Recurrent
Cases	9 (Onset definitely occurred during night)	5	1 (Gradual onset with colds; cleared up as colds improved)

TABLE 6.—*Roentgen Observations on the Nasal Sinuses and Optic Canals* *

	Clouding of Ethmoids	Clear Sinuses	Large Sphenoid Sinus
Cases	5	10	15

* The optic canals were uniformly measured in the last nine cases (those seen since 1924); in several severe cases large or average-sized canals were found.

TABLE 7.—*Gross Pathologic Observations on the Sinuses at Operation in Nine Cases (Three Bilateral)* *

	Normal	Polypi, Pus, Inflammation, etc.
Cases	8	1

* The operation consisted of submucous resection of the nasal septum, posterior ethmoidectomy and opening the sphenoid sinus. The presence or absence of pathologic changes in the sphenoid sinus was determined by the inspection of the interior of the sinus by means of a nasopharyngoscope passed through the operative opening.

I believe that the size of the optic canal does not determine the vulnerability of the optic nerve. In my opinion, this is determined by the structure of the wall between the optic nerve and the sphenoid or posterior ethmoid sinuses.

Herzog, as already mentioned, found in microscopic sections a marked difference in the structure of this bony wall. Some of the walls had a dense, nonporous structure; others, large spongelike marrow spaces. In the marrow spaces he found a union of the deeper layers of the submucosa of the nasal sinuses and cellular elements given off from the dural sheath of the optic nerve.

10. White, Leon: An Anatomical X-Ray Study of the Optic Canal in Cases of Optic Nerve Involvement, Boston M. & S. J. **189**:741 (Nov. 15) 1923.

When I read his work, I was immediately impressed with the great possibilities of this fact as explaining the mechanism of the production of retrobulbar neuritis originating in the nasal sinuses, and it seemed to me that if a method could be found which would demonstrate on the living patient the thickness of the bony wall between the optic nerve and the sphenoid sinus, such a method might prove valuable.

My method of determining the thickness of the bony wall between the optic nerve and the sphenoid sinus is to fill the latter with a radiopaque oil, using a blunt cannula inserted through the ostium, and to place the patient so that the oil gravitates to the upper outer angle of the sphenoid sinus. With the head in this position, the roentgen rays are passed through the skull in the same plane as that described by Leon White in his roentgenologic study of the optic canals.

ROENTGEN TECHNIC

Dr. Sidney Lange's roentgen technic for the Vail-Lange visualization of the wall between the sphenoid sinus and the optic canal is:

Right Sphenoid Sinus.—After iodized poppy seed oil 40 per cent has been introduced into the right sphenoid sinus, the patient lies on a flat table either prone or partially on the right side (if he has a short rigid neck). A pillow or sandbag is placed under the upper part of the chest to elevate the shoulders slightly, so that the head can be angulated downward (flexed) slightly.

A cassette, 8 by 10 inches (20 by 25 cm.), is placed at an angle of 45 degrees and the right side of the face is placed obliquely against it so that the right malar prominence, tip of the nose and point of the chin are in contact with the cassette.

The axis of the x-ray beam is directed through the left parietal eminence and through the center of the right orbit, exactly at right angles to the plane of the cassette.

Using 20 ma. at a 4½ inch spark gap, the time of exposure would be two seconds.

Left Sphenoid Sinus.—The position of the head is reversed.

SUMMARY

1. A brief résumé of the literature quoted by Canuyt, Ramadier and Velter would seem to show that retrobulbar optic neuritis of nasal origin has been recognized for several centuries.

2. The pathologic studies of Professor Herzog, of Innsbruck, are quoted, and his belief is emphasized that they will explain in every way the mechanism of the production of retrobulbar optic neuritis from posterior nasal sinusitis.

3. Statistics of various authors are quoted to show that the incidence of retrobulbar optic neuritis originating in the nasal sinuses varies according to the particular author's acceptance or denial of a nasal sinus etiology of retrobulbar optic neuritis.

4. A series of fifteen cases of retrobulbar optic neuritis of nasal origin seen by me are tabulated.

5. An important observation is made, namely, that when the onset of blindness was acute and could be definitely dated, it was always noticed after awakening from sleep. The position of the head in sleeping favors the gravitation of infection to the upper outer angle of the sphenoid sinus where the optic nerve is in closest relation.

TABLE 8.—*Final Visual Acuity*

	No Operation (6 Cases)		Operation (9 Cases)		
	No Treatment	Recovery; Vision 20/25 or Better	Complete Recovery; Vision 20/20	Vision 20/30	Slight Improvement; Vision 20/100 or Less
Cases.....	3*	3	7†	1	1‡
Per cent.....	50	50	77	11.1	11.1

* One of these patients (the recurrent case) would not consent to operation.
† This includes one of the bilateral cases; vision in one eye was 20/20; in the other, 20/100.
‡ This does not include one of the bilateral cases; vision in one eye was 20/100; in the other eye, 20/20.

6. Statistics are quoted to show that the ocular symptoms constitute an early indication of multiple sclerosis in only 14 per cent of the cases, and the opinion is stressed that the virus of multiple sclerosis may gain entrance to the central nervous system from the sphenoid sinus in the same way that organisms have been shown to do. If this is a fact, it would immediately indicate that an early operation on the posterior sinuses should be performed in cases of multiple sclerosis.

7. A new method is described of roentgen visualization of the relations between the optic canal and the sphenoid sinus by means of filling the sinus with radiopaque oil and so placing the patient's head that the upper outer angle of the sinus is dependent. Thus, the radiopaque oil comes into contact with the boundary of the sphenoid sinus where it is most closely related to the optic nerve. In no other position can this be shown.

Clinical Notes

MALIGNANT NEUTROPENIA (AGRANULOCYTOSIS)

Report of a Case with Apparent Recovery *

E. G. Gill, M.D., Roanoke, Va.

The term agranulocytosis [1] has come into the literature because of its brevity. The name is not strictly correct. "Agranulocyte" was the term originally chosen for neutrophils in leukemia without granulations, and by the term "agranulocytosis" is meant an increase in these atypical neutrophils; this was not intended. The increase of monocytes accompanying this extreme injury of neutrophils is important, a fact that speaks against Naegeli's myelogenic genesis. The bone marrow is degenerated to a great extent; it is liquid, with few cells, most of which are plasma cells and lymphocytes, and it is practically without agranulocytes.

Since similar blood pictures have been obtained experimentally in anaphylaxis (by myself and Miss Loewe), it seems possible that agranulocytosis is an anaphylactic condition instead of an individual disease. In the anamnesis of the condition, cases of angina, grip, etc., are often found. At present, the name applied by Tuerk, "malignant neutropenia," seems to be more appropriate.

The syndrome of agranulocytosis with angina is threefold: [2] (1) an acute systemic infection with prostrating fever; (2) a markedly low number of white cells and greatly diminished or absent polymorphonuclears; the other blood elements, including the platelets, relatively normal, and (3) ulcerative and necrotic lesions of the oropharynx, including the lips, gums, tongue, pillars, tonsils, pharynx and sometimes the intestinal tract and genital areas.

The condition appears at any age, and the majority of cases occur in women. It has no significant racial, geographic, epidemic or contagious incidence. In about half of the cases reported, jaundice developed as the disease progressed.

It is not known whether this condition is due to a congenital deficiency in the bone marrow and an endocrine imbalance of the spinal cord function, or to a complete paralysis of function as expressed by Zilowsky: [3] "sepsis in a weakened organism resulting in an injury to leukopoietic system, paralysis of the organ complex, liver, spleen and endocrine secretions which stimulate the bone marrow and regulate the entrance of granular cells into the blood stream." The essential pathologic changes appear in the red bone marrow. In agranulocytosis, the portion of the marrow that produces agranulocytes is completely suppressed—in normal conditions, the life of this cell is from three to five days—while the portion of the marrow that produces the erythrocytes and platelets continues to

* Submitted for publication, March 18, 1931.

1. Schilling, Victor: The Blood Picture and Its Clinical Significance (Including Tropical Diseases). A Guidebook on the Microscopy of Blood, St. Louis, C. V. Mosby & Company, 1929.

2. Babbitt, J. A., and Fitz-Hugh, T., Jr.: Agranulocytic Angina; Report of a Case with Apparent Recovery, Arch. Otolaryng. **12**:439 (Oct.) 1930.

3. Zilowsky, quoted by Babbit and Fitz-Hugh (footnote 2).

function normally. It is not known why one portion of the bone marrow should be paralyzed while the other continues to function normally. It is also not known whether the pathologic condition in the bone marrow precedes or follows a local infection. Certainly, in some cases, the patient is ill before the development of local lesions is detected, but how is one to account for the condition of the blood marrow?

Vincent's organisms are frequently found in cultures taken from the ulcerative lesions, but not to an extent that would prove them to be a definite etiologic factor. Cultures of the blood fall in the same category. Some physicians have attached significance to the fact that many attacks of agranulocytosis have been preceded by shock, including fractures of the bone with accompanying cellulitis, by the extraction of teeth, the administration of neoarsphenamine, vaccination for typhoid, arthritis and the removal of the thyroid gland.

The condition frequently passes for diphtheria, Vincent's angina, syphilis or a streptococcic sore throat. An examination of the blood, however, will establish the diagnosis, and in cases with ulcerative lesions of the mouth and throat, careful smears, cultures and examinations of the blood should be made.

Many different types of treatment have been employed. The best results seem to follow repeated transfusions of blood, stimulating irradiation over the long bones and local treatment for the lesions of the mouth and throat, combined with a supportive diet rich in vitamins.

In the following case apparent recovery from malignant neutropenia occurred.

A farmer, aged 57, had been in good health until Nov. 20, 1930, when he began to have chilly sensations which continued for three days. On the fourth day, the chills were severe and frequent, and at this time severe pain developed in the entire left side of the jaw and in the face and temporal region. The condition grew progressively worse until his admission to the hospital on December 10, when he presented an appearance of extreme prostration.

His chief complaint was severe pain in the left side of the jaw and in the face and temporal region. On admission, the temperature and pulse rate were normal. Six hours later he had a chill that lasted for twenty minutes, and his temperature rose to 104 F. Physical examination gave negative results, except for jaundice, which was definite. The paranasal sinuses were clear. There were two small, superficial ulcers on the margins of the gums, smears from which showed *Staphylococcus aureus*. On admission, the examination of the blood showed: coagulation time, one minute; white cells, 1,950; red cells, 4,320,000; hemoglobin, 90 per cent; polymorphonuclear neutrophils, 18 per cent, and lymphocytes, 82 per cent.

The patient was given a transfusion of 500 cc. of whole blood, without any reaction. The pain in the left side of the jaw and face was immediately relieved. For two days his condition improved. On the third day following the transfusion of blood, there was marked pain in the jaw and face, and his general condition was worse. An examination of the blood after the transfusion showed: white cells, 4,900; red cells, 4,500,000; polymorphonuclear neutrophils, 36 per cent; lymphocytes, 57 per cent; eosinophils, 5 per cent; basophils, 3 per cent, and lymphoblasts, 1 per cent. Four days following the first transfusion of blood, 500 cc. of whole blood was again given, without reaction. The patient's general condition improved immediately and continued to do so. On December 23, he was discharged from the hospital free from symptoms and with a normal blood count.

SUMMARY

As a recurrence of malignant neutropenia should always be considered, this case is reported as one of apparent recovery. When seen two months after leaving the hospital, the patient was well and attending to the duties of his farm. An examination of his blood showed that it was normal.

The most striking features of this case, other than the blood picture, were the extreme prostration, marked jaundice and severe pain in the left side of the jaw and in the face. The treatment consisted of two transfusions of blood and large doses of cod liver oil and glycerite extract of red bone marrow by mouth. With few exceptions, the course and prognosis of this disease are usually short, severe and fatal.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

---

TONSILS AND ADENOIDS

FRENCH K. HANSEL, M.D.
ST. LOUIS

In reviewing the literature for 1930 on the tonsils and the adenoids, I have selected only those papers which promote discussion and those which, according to my judgment, show the advances made in this particular field of otolaryngology during the past year. Interesting papers by Monroe and Volk, McLean and Kaiser were directed toward the study of large groups of children for the results of tonsillectomy. A great deal of interest has been exhibited in the consideration of the complications of tonsillectomy. A symposium on the subject of pulmonary abscess following tonsillectomy, which was given before the American Bronchoscopic Society, considered all phases of this subject in a comprehensive manner. Other complications of tonsillectomy, such as thrombophlebitis of the internal jugular vein, pyemia, deep cervical infection and parapharyngeal abscess, were presented by various authors in several interesting papers. In the treatment of the tonsils, renewed interest has been exhibited in the use of electrocoagulation and irradiation. Several new instruments have been designed and described.

GENERAL CONSIDERATIONS

Monroe and Volk analyzed the complaints of 736 children before and after operation, and compared the results with an analysis of the complaints of 741 children in a control group having similar symptoms on examination and reexamination one year later. The following conclusions are drawn: Tonsillectomy offers a child considerable relief from such common conditions as sore throat, head colds and breathing through the mouth. It reduces malnutrition and promotes the disappearance of enlarged cervical glands. Complaints have been relieved in 91 per cent of patients operated on. In order to obtain the maximum benefit, the child should be operated on in early childhood. Improvement followed operation in 40 per cent of children with subnormal mentality and showing retarded progress in school resulting from enlarged tonsils and adenoids. In the 736 cases analyzed, coexisting conditions were present in 120. The control group of cases did not

show any improvement during the one year of observation, as compared with 91 per cent of improvement in the group of patients operated on.

In an article on the incidence of recurrent respiratory infections in childhood, McLean stated that recurrent respiratory infections were prevalent in both the patients who had had the tonsils and adenoids removed and in those who had not undergone the operation. Of the children under 3 years of age with recurrent respiratory infections, only 6 per cent had had the tonsils and adenoids removed. Of the children over 3 years of age with recurrent respiratory infections, 49 per cent had had the operation. The variation in the percentage of the recurrent incidence, according to ages, in children who had had tonsillectomy and adenoidectomy can be explained by the fact that many children over 3 years of age have had the operation, while very few of the younger children have had the tonsils and adenoids removed.

After a comparative study of 2,200 tonsillectomized children with an equal number of controls three and ten years after operation, Kaiser reported the following interesting conclusions:

1. The real value of the removal of tonsils and adenoids cannot be definitely established in a few years. Apparent benefits during the first few postoperative years are not so evident over a ten year period.

2. Outstanding benefits are apparent in influencing the incidence of sore throats over a ten year period.

3. Substantial benefits are apparent in rendering children less susceptible to scarlet fever and diphtheria.

4. Acute head colds and otitis media, though definitely lessened over a three year period, are not essentially influenced over a ten year follow-up period.

5. Cervical adenitis is decidedly reduced in tonsillectomized children over a ten year period.

6. The respiratory infections, such as laryngitis, bronchitis, and pneumonia, not only are not benefited but actually occur more frequently in tonsillectomized children.

7. First attacks of rheumatic manifestations occur from 30 to 50 per cent less often in tonsillectomized children. The greatest reduction occurs in children tonsillectomized early. Recurrent attacks are not benefited at all.

8. Incomplete tonsillectomies do not offer the same protection against the usual throat complaints and infections as complete removal of tonsils.

9. The hazards of tonsillectomy must be considered in evaluating the end-results. Considering this hazard, the late results seen in 2,200 children ten years after operation are evident only in the reduction of sore throat, cervical adenitis, otitis media, scarlet fever, diphtheria and rheumatic fever and heart disease.

In a statistical study of the children in public schools for the deaf, Shambaugh, Hayden, Hagens and Watkins reported on the relation of tonsils and adenoids to lesions of the middle ear. Among the children examined, 2,355, or 44.03 per cent, had their tonsils and adenoids removed, 2,638, or 49.32 per cent, still had the tonsils and for 355 no

record was made regarding the tonsils. Of the children who had had the tonsils and adenoids removed, 1,377 showed no palpable evidence of having had disease of the middle ear. Of the children with tonsils still in situ, 1,062 showed palpable evidence of disease of the middle ear and in 1,576 such evidence was absent. Disease of the tonsils and adenoids can be responsible for severe deafness only when it has produced otitis media which has invaded the labyrinth—a negligible factor in these statistics.

From a clinical and roentgenologic study of 174 children with hypertrophy of the tonsils necessitating operation and of 100 children without enlargement, Hofer concluded that there is no connection between tuberculosis of the tracheobronchial lymph nodes and hypertrophy of the tonsils. He did not favor the theory propounded by many authors that enlargement or tuberculosis of the tracheobronchial lymph nodes with coincident hypertrophy of the pharyngeal tonsils is due to a descending infection of the lymphatic system. Hypertrophy of the pharyngeal tonsils is related to tuberculosis in children so far as inhibition of nasal breathing leads to a chronic bronchitis, which, according to statistics, is responsible for concurrent infection by tuberculosis in 95 per cent of cases. Very few cases are known in which tuberculosis of the pharyngeal tonsils is primary to tuberculous lymph nodes. In four fifths of Hofer's cases hypertrophy of the pharyngeal tonsils was coincident with an enlargement of the cervical lymph nodes. Hypertrophy of the palatine tonsil was almost invariably attended by hypertrophy of the cervical lymph nodes. Enlargement of the cervical glands without hypertrophy of the tonsils may be referred to scrofula and lymphatic diathesis. If indications for adenotomy, tonsillectomy or tonsillotomy are given, these operations should be performed, according to Hofer, in spite of a positive cutireaction for tuberculosis and positive results in roentgen examination. The effect of the operation on the pulmonary condition is invariably favorable and may be continuous.

In the literature on the subject of tonsils and adenoids reviewed it is remarkable that little if any attention has been paid to the adenoids. In an interesting and comprehensive report on nasopharyngeal abscess, Yankauer reviewed his series of 155 cases. He pointed out the difference between nasopharyngeal abscess and Thornwaldt's disease. He referred to Thornwaldt's original report, an analysis of which showed that the latter's cases were cases of nasopharyngeal abscess and not pharyngeal bursitis. Nasopharyngeal abscess arises from the recessus medius of the adenoid. Yankauer has devised a direct speculum for examination of the nasopharynx. The largest number of cases occurred in patients between the ages of 20 and 30 (72 of 155 cases); 32 cases occurred in persons between the ages of 30 and 40. An

abscess in the adenoid is characterized by the presence of a swelling in the region of the recessus medius, but the swelling may not manifest itself as a rounded protuberance, but merely as an elevation of the concavity of the vault of the nasopharynx, a flattening out of the concave surface. The swellings do not always contain pus and the diagnosis is not complete until the swelling is opened and the contents are noted. Of 142 cases in which the contents were noted, 121, or 86 per cent, showed thick creamy pus. Yankauer described the symptoms produced by these abscesses. It is particularly interesting that headaches may be caused by these infections. Yankauer's interesting report should prompt the rhinologist to be more careful in the examination of the nasopharynx to determine the presence or absence of nasopharyngeal abscess.

Bradley has made an attempt to assess the value of the tonsil-adenoid operation in the prevention of epidemic nasopharyngeal infections. The operation was found to be of no appreciable value, but some evidence was produced to show that it is followed by compensatory hypertrophy of other lymphoid tissue in the presence of acute disease of the upper passages. The conditions found suggest that, until the loss of lymphoid tissue is made good, patients wo have been operated on are more susceptible to the common catarrhs and more frequently attacked by complications. Chronic tonsillitis is a complication of acute nasopharyngitis. Autogenous reinfection from septic tonsils is not common. It is discussed and the necessity for operation in these circumstances emphasized. A distinctive type of epidemic sore throat is described. Tentative statistical proof is presented that coryza and febricula are "formes frustes" of the stationary fevers in a community.

Miller discussed disease of the lingual tonsil and called attention to the fact that it is frequently overlooked in examination of the throat. Hypertrophy is the most common disease, but acute and chronic infections occur with fair frequency. The incidence of involvement seems greater in men than in women, and may occur in infancy. The best treatment for chronic involvement of the lingual tonsil is surgical removal, though the use of the galvanocautery is of decided value.

It was suggested by Jerwood that maternal mortality might be reduced by eradicating focal sepsis, preferably before pregnancy has occurred. The tonsils are the commonest site of focal sepsis in young adults, and therefore in most pregnant women. Just as influenza and nasopharyngeal catarrhs constitute an overwhelming majority of the acute illnesses encountered in general practice, so chronic sepsis of the upper respiratory tract lies at the root of most chronic debilities.

Snoke reported a case of lympho-epithelioma involving both tonsils. On first examination the left tonsil only was involved. This was treated with radium. Two years later the right tonsil became involved. At this time nodules were noted at the inner canthus of the left eye in the

pharynx and nasopharynx. Pathologic examination of the tonsils showed a lympho-epithelioma. Unfortunately, blood counts were not recorded in this case. Tumors of a similar nature were reported a few years before by New in cases of chronic lymphatic leukemia.

ANATOMY AND PHYSIOLOGY

The surface of the tonsils is not indicative of their pathologic condition, according to Heiberg. It is only by microscopic examination of the adenoid tissue that the severity of infection of the organism may be inferred and a guide in prognosis found. The centers of phagocytic activities (or germinal centers) especially show variations of structure which have been found to correspond to the degree of involvement. They are larger in the healthy organism, which indicates a metabolic relation. Heiberg referred to his previous papers on this subject. In a study of 100 cases in which operation was performed at the Finsen Institute, Heiberg did not find any difference in the behavior of pharyngeal and palatine tonsils. The activity of the germinal centers, according to Heiberg, appears to lie in the direction of the destruction of lymphocytes, whereas production of the latter takes place in other parts of the tissue. The lymphocyte cap surmounting the germinal centers is larger if these are defective, and several distinct types may be distinguished in their organization. The structure of the lymphocytic nuclei is more highly differentiated in the adenoid tissue than in the lymphocytes of the blood or in those seen in infiltrations surrounding the blood vessels in acute inflammations. Greater development of the interstitial tissue is found in infections. However, the rôle of immunity and metabolism is not yet clearly defined. Heiberg pointed to the substance that was isolated by von Schmidt, in 1927, from the germinal centers of the tonsils. It was found to be thermostabile in acids after removal of the proteins, and it caused lymphopenia after subcutaneous injection. Stimulation of the tonsils by "massage" has likewise caused lymphopenia.

In a study of the position of the deeper vessels of the neck in relation to the end of the needle in local anesthesia around the tonsil, Stout concluded that the large vessels of the neck are sometimes in close proximity to the tonsil. The usual type of needle used is sufficiently long to reach these vessels. Veins are more easily pierced than arteries, and sharp needles enter the vessels more easily than round pointed ones. The lack of resistance encountered in injections of a local anesthetic suggests the likelihood of injection into a vessel. This may account for some of the marked symptoms that sometimes follow the injections.

BACTERIOLOGY AND PATHOLOGY

In 400 tonsillar cultures examined by Cobe, staphylococci were the predominating organisms. The streptococci followed the pneumococci

in predominance, with hemolytic streptococci the principal members of the group. Three per cent of the nonhemolytic streptococci recovered were classed as *Streptococcus cardio-arthritidis*. There appears to be a definite relationship between the type of organism recovered from tonsillar cultures and the age of the patient, streptococci being more common in younger patients (under 11 years of age). There was a definite seasonal difference in the organisms recovered from tonsillar cultures, *Bacillus influenzae, Bacillus mucosus-capsulatus* and the diphtheroid types being more prevalent in the spring, and *Micrococcus catarrhalis* more prevalent in the fall.

Brandes' experiments were made with toxins of streptococci isolated from five patients with acute articular rheumatism and from a patient with chronic articular rheumatism. Twice the condition was associated with acute tonsillitis, twice the patient's history revealed recurring sore throat, and twice chronic tonsillitis was present. Liver bouillon was used for cultures; it contained 1,000 cc. of liquid, 500 Gm. of liver, 12.5 Gm. of a proprietary dry peptone and 5 Gm. of sodium chloride. After sterilization, a 30 per cent solution of sodium carbonate was added until red litmus paper became slightly blue. The central portion of the tonsils was repeatedly washed in a physiologic solution of sodium chloride and then ground into a pap which was cultivated on the bouillon and on blood agar. The culture tubes were kept in the incubator for from one to thirty-five days. From 3 to 31 cc. of the culture was introduced into the marginal auricular vein of rabbits at once or in repeated injections. In thirteen of the thirty-nine animals thus inoculated, lesions developed in the joints; there was a seromucous exudate which contained polymorphonuclear leukocytes and an increased number of large mononuclear cells. The changes in the joints resembled those in cases of rheumatic polyarthritis in man, but the swelling of the joints was never pronounced. The results suggest that toxins of hemolytic streptococci may possibly be the causal factor of articular rheumatism in man.

Castellani said that diseases of the tonsils due to fungi are not rare, though so far scant attention has been paid to them. They may be classified in various ways. Castellani presented a clinical classification as probably the most useful for practitioners. According to this classification tonsillomycoses are separated into two groups, acute and chronic. Of the acute tonsillomycoses the principal types are tonsillomycosis follicularis and tonsillomycosis membranacea (tonsillomycosis diphtheria-similis). Cases of the latter type may closely resemble those of diphtheria. Of the chronic and subacute tonsillomycoses the principal types are tonsillo-actinomycosis, tonsillopseudo-actinomycosis, tonsillomycosis fusca and tonsillomycosis spiculata vel spinulosa.

By the use of penicillin, hemoglobinophilic bacilli were isolated by Fleming and MacLean from the gums of 30 healthy nurses and students

and from the tonsils and postnasal spaces of almost all of these. The majority of the strains isolated were nonhemolytic, but hemolytic strains were found in all situations. Para-influenza as well as influenza bacilli were isolated from all these situations. A bacillus agreeing in its (growth) requirements with *Bacillus haemoglobinophiluscanis* was isolated from the gums of 1 person. From the gums of 6 laboratory workers who were studied carefully, both influenza and para-influenza bacilli were isolated. The hemoglobinophilic bacteria isolated were variable in their cultural and morphologic characteristics. A type of para-influenza bacillus that was constantly found in the gums was described. From the apexes of infected teeth, hemoglobinophilic bacilli were isolated in 3 of 4 cases. The methods of using penicillin for the isolation of influenza bacilli are described.

Castellani discovered that fetor oris of tonsillar origin is always associated with the presence of two bacilli, for which he proposed the names *Bacillus colofoetidus* and *Bacillus alkalofoetidus.*

In an article on mixed chancre of the tonsil, Vialle, LeCocq and Ronchèse reported two cases in which Vincent's spirillum and the fusiform bacillus attacked syphilitic lesions of the tonsil, causing lesions that simulated neoplasms and that were of extreme discomfort and danger to the patient. They suggested the desirability of tests for syphilis in all ulcerative lesions of the tonsil, no matter what the smear or dark field may show.

In a study of the pathogenic tonsil Guttman found that by gentle massage of the tonsils certain patients revealed an increased white blood cell count while others showed a decreased count. In attempting to standardize the results of this manipulation, he came to the following conclusions: 1. Massage of the empty fossae of tonsillectomized patients causes little or no change in the white count of the peripheral blood. 2. Hyperplastic tonsils, when not accompanied by infection, cause a fall in the white count. 3. Massage of a chronically infected tonsil causes a rise in the white cell count, thus serving as a basis for a biologic reaction from which the pathogenic status of the tonsil may be determined. A healed and noninfective tonsil will not react in this manner. 4. The test is contraindicative in the presence of an acute or a subacute process in the tonsils or in a systemic disturbance that may be predicated on a tonsillar infection.

COMPLICATIONS

During the past year a number of interesting reports have appeared on the subject of pulmonary abscess following tonsillectomy. In a symposium Clerf, Carmody, Pierson, Holman and Schall discussed the various aspects of the subject. Clerf reported a group of 77 cases. Fifty, or 65 per cent, occurred in patients between the ages of 20 and 40. This corresponds rather closely to the age-abscess incidence of pulmonary

abscess in all cases. In 4 cases local anesthesia, and in 73 cases general anesthesia, was used. In 48 cases (62 per cent) the right lung was involved, and in 28 (37 per cent) the abscess occurred in the left lung; in 1 case the lesion was bilateral. The underlying principles in the treatment for pulmonary abscess, irrespective of the method to be employed, consist of the establishment and maintenance of adequate drainage. Clerf advocated bronchoscopy early in the course of the disease; 38 patients were ultimately discharged as well after a course of bronchoscopic treatment. The average duration of the abscess before treatment was four and one-half months. In 27 cases the duration was three months or less; in 5 cases it was more than one year. No definite rules can be laid down with regard to the length of time bronchoscopy should be continued; every case of abscess of the lung should be considered individually by the internist, roentgenologist, surgeon and bronchoscopist to determine the form of treatment to be employed.

In discussing the laryngologic aspect of pulmonary abscess following tonsillectomy Carmody gave a brief but interesting history of infection of the lung as being derived from the upper respiratory tract. He called attention to the fact that textbooks on diseases of the nose and throat give little consideration to this subject. The theories of origin of pulmonary abscess were considered. The embolic and aspiratory routes were described. Bronchoscopic evidence points to aspiration of septic material from the upper respiratory tract as the most common cause.

In the consideration of the medical aspects of posttonsillectomic pulmonary abscess Pierson defined this type of condition as characterized by consolidation and necrosis. The complication generally begins from twenty-four to forty-eight hours after operation, and is characterized by fever, sweats, anorexia, malaise and a dry harassing cough terminating, in from ten to fourteen days, with foul expectoration. In summarizing the medical aspects Pierson emphasized the following points:

> Pulmonary abscesses follow, although not frequently, operation on the upper respiratory tract and teeth, and may be produced by aspiration or emboli. The anaerobes may be of particular importance in the formation of abscesses in otherwise merely pneumonic processes. Physical signs are less helpful than a carefully secured history and a series of roentgenograms in the diagnosis and management of the disease. The bronchoscopist and surgeon are most important members of the team, and should frequently be in consultation with the internist. Medical treatment offers a much brighter outlook per se and as a preparation for operation than was the case in early surgical procedures. Some abscesses may be prevented by thorough examination of the patient to rule out acute and localized pulmonary disease and by more attention to hygiene of the mouth before and after operation.

In discussing the factors of healing in posttonsillectomic pulmonary abscess Holman stated:

> As knowledge in the treatment of patients with intrapulmonary abscess is gained and recorded, it becomes increasingly evident that there are various routes

which may be, and which sometimes must be, traversed before ultimate recovery from such an abscess is achieved. The most favorable time for the cure of the abscess is during the period immediately following its inception, and the treatment then will necessarily be different from that attempted at a later stage when the thickened, rigid walls of the abscess present one of the most baffling problems in surgery.

As to its development, an intrapulmonary abscess may be produced by the destruction and liquefaction of pulmonary tissue through the action of infecting organisms. These organisms may reach the lung by way of the bronchi or the blood stream.

Whatever may be the origin of the infecting organism, whether through the blood stream or the bronchi, the resulting destruction of pulmonary tissue leaves a dead space—the abscess cavity—which must be obliterated. The three important factors in the obliteration of this cavity are: (1) adequate drainage, (2) the gradual contraction of fibrous tissue laid down as granulations in the wall forming the abscess and (3) the expansion of the healthy normal lung to replace the destroyed lung.

Bronchoscopic considerations as an aid to the surgeon are presented by Schall, who concluded that the bronchoscopist is of aid to the thoracic surgeon in localization of the abscess (1) by following the pus stream to its source and (2) by pneumography. The bronchoscopist can improve drainage by the dilation of strictures and by the removal of granulation tissue that may obstruct the bronchus. In certain cases, with bronchoscopic sounding the surgeon can open the abscess by cutting down on the bronchoscope.

Hara reported the result of the bronchoscopic study of 110 tonsillectomized patients for evidence of aspiration. He strongly emphasized the desirability of operating on these patients in the modified Trendelenburg position as a means of preventing aspiration.

In a discussion of the complications of tonsillectomy Hartz gave a comprehensive review of the various important factors to be considered. He emphasized the importance of a complete history and physical examination, and advocated that plenty of time should be allowed for the subsidence of acute infection before tonsillectomy was performed. A routine laboratory procedure for testing the coagulation of the blood, roentgenograms of the chest in suspected cases of disease of the thymus gland, the control of hemorrhage and postoperative care are also emphasized.

According to Dixon and Helwig, thrombophlebitis of the internal jugular vein probably takes first rank as a fatal complication of tonsillitis. These authors reported a group of 8 cases resulting from an extension of an acute and overwhelming infection originating in the tonsil. Trauma to the inflamed tonsillar or peritonsillar tissues pre-

disposes to this complication. In most cases the phlebitis occurs as a complication of the primary attack of tonsillitis, which would indicate a lack of local resistance and a lowered immunity to the infecting organism. Resection of the infected vein and removal of the thrombus are always indicated when a positive diagnosis can be made early enough to warrant intervention.

Goerke directed attention to the fact that a tonsillectomy that is performed to prevent pyemia occasionally may lead to it. He reported a case from his own observation. In a girl, aged 20, who had frequent attacks of tonsillitis, a tonsillectomy was performed ten days after an attack, and pyemia developed. It was the author's opinion that this complication set in because the interval between the tonsillitis and the tonsillectomy was too short. He emphasized that from four to six weeks should elapse after a tonsillar infection before a tonsillectomy is performed. This precaution had been disregarded in this instance because the patient did not wish to lose too much time. In order to prevent infection spread by the needle that is employed for the administration of the local anesthetic, the author advised that several needles be kept in preparation, and that if one penetrated into the tonsillar tissues it should be exchanged for a sterile one. As another prophylactic measure, he emphasized that all carious teeth should be removed or treated before the operation is begun. In order to detect possible latent infections, the temperature should be measured during the last days preceding the tonsillectomy. During the operation itself, sterilization of the hands and instruments is not sufficient. All unnecessary tearing of the tissues should be avoided. Frequent dabbing may also have harmful effects; it will not be necessary if, after the separation of the upper pole, a tampon is placed in the upper tonsillar niche. This tampon should remain until the operation is completed. Tamponade after completion of the surgical intervention is, according to the author's experience, of no significance for the prevention of postoperative infection. However, he advised that the wound be covered with an antiseptic iodine powder, which also has a hemostatic effect. In regard to the treatment for tonsillogenic pyemia, which may have been caused either by tonsillitis or by tonsillectomy, the author stressed the following precautions: Early intervention is necessary. If attacks of chills recur and if other causes may be excluded, treatment should be instituted at once. Cervical mediastinotomy, with exposure of the jugular vein, ligation of the vena fascialis communis and drainage of the parapharyngeal space is recommended. If the facial vein is thrombosed, either excision should be done or the jugular vein should be ligated above and below the place of anastomosis of the vena fascialis communis. If the venous thrombosis is extensive, the thrombosed vessels should be removed following the ligation of the jugular vein. If the jugular vein

itself is diseased, ligation should be done as deeply as possible, the vein should be slit or excised, and ligation should also be done at the base of the skull. If the jugular vein is not involved, it should be let alone.

Thirty cases of deep cervical infection following tonsillectomy, including 3 fatalities, are reported by Shapiro and 80 cases are collected from the literature. In 94 per cent of all the cases reported, the operation was done under local anesthesia. The most important factor in the etiology is the injection of infected solution into the parapharyngeal space. Two clinical varieties are noted: a phlegmonous type, which includes a large majority of all cases analyzed, and a vascular infection, which manifests itself in the form of septicemia, thrombosis or embolism. The phlegmonous form of deep cervical infection following tonsillectomy is characterized by a symptom-complex consisting of trismus, fever and swelling of the neck on the affected side. A large number of grave additional complications are recorded. Shapiro urged that when local anesthesia is used, three points are essential in the prophylaxis: a proper technic of injection, the avoidance of contamination of either the needle or the solution and a minimum period of four weeks between the last acute attack and the operation. The treatment should aim at localizing the infection before an attempt is made to drain the abscess. The incision of choice is through the tonsillar fossa. External operation is indicated under certain conditions.

Weidlein reported an interesting case of chronic abscess of the pharyngomaxillary fossa without symptoms. A deep pocket was present in the pharyngomaxillary region, the outlet of which was discovered accidentally during an operation for the removal of tonsils and which apparently gave no symptoms referable to its presence. The patient had had many previous attacks of peritonsillar abscess. After tonsillectomy the tract was shown roentgenologically by means of the probe and injections of iodized poppy seed oil 40 per cent. In the differential diagnosis a bronchial cyst and abscess were considered. The likelihood of a cyst being present was ruled out on the basis of the fact that there was no discharge from the tract following the operation on the tonsils. It was concluded that the cavity represented an old abscess, probably of tonsillar origin.

Watson-Williams reported 7 cases of parapharyngeal abscess which closely resembled peritonsillar abscess. The most striking difference is the absence of any form of edema of the palatal mucosa. The condition should be suspected when an attack of "quinsy" appears to run an atypical course, or when incision in the usual site on the tonsil fails to reveal pus. The abscess tends to point low in the side of the pharynx behind the posterior faucial pillar where it is permissible to open it under gentle pressure with a blunt instrument.

Two interesting cases of infection involving the pharyngomaxillary space which originated in the tonsils were reported by White. One patient died of meningitis from intracranial extension, and the other recovered after external drainage of this space. White therefore advocated external drainage as recommended by Mosher.

For the alleviation of pain in peritonsillar abscess Guttman described a method that involved cocainization of the palatine nerves passing through the sphenopalatine ganglion. A topical application of cocaine is made on the lateral wall just posterior to the posterior tip of the middle turbinate. In this manner pain may be controlled and incision of the abscess may be performed with little discomfort to the patient.

Von Hofe reported 3 cases of emphysema of the head and neck complicating tonsillectomy. It seems possible that this condition may be brought about as follows: 1. Air may enter the tissue following perforation of the tonsillar fossa bed. 2. Air may enter the tissues following perforation of pulmonary vesicles. 3. Air may enter the tissues after being forced into Wharton's duct and thence diffused.

A rare complication in a case reported by Hochfilzer was that of phlegmon of the neck and osteomyelitis of the mandible following tonsillectomy.

An interesting report on *Bacillus proteus-vulgaris* infection of the mastoid in 1 case and of the tonsils in 2 cases calls attention to the fact that this infection occurs in the ears and throat as well as elsewhere in the body. The characteristics are a slow, progressive putrefactive necrosis, extreme toxemia and an extraordinary loss of weight. The treatment calls for early drainage and exposure, frequent cleansing and the use of antiseptics rich in oxygen. The prognosis depends on the site of invasion, the virulence of the organism and the individual resistance, but the infection is usually a terminal one.

Ballenger reported 2 cases in which appendicitis developed soon after the removal of the tonsils. A girl, aged 5, was admitted to the hospital with a history of breathing through the mouth, snoring, frequent colds, sore throats and an occasional earache, with two instances of a supervening acute suppurative otitis media. There was no history of a previous attack of appendicitis, a recent cold in the head or sore throat. The tonsils were large and almost met. No acute inflammation of the nose or throat was evident. The tonsils were removed. The second day following the operation, the patient suddenly complained of pain in the abdomen. The pain was accompanied by a rise in temperature to 101 F., and a leukocyte count of 18,000. A diagnosis of acute appendicitis was made, which was confirmed at operation. The second patient, aged 5 years, was admitted to the hospital with a history of frequent colds and sore throat, and of breathing through the mouth and snoring most of the time during the past year. The tonsils and adenoids were

large, with no evidence of an acute infection and with no history of a recent cold or sore throat. The tonsils were removed. The day following tonsillectomy she felt well, but during the night of the following day she complained of pain in the abdomen and nausea. Vomiting and fever accompanied the pain. A diagnosis of acute appendicitis was made. The abdomen was opened, and a gangrenous appendix was found with localized peritonitis.

Hyperpyrexia, fatal or not, is a most unusual complication following removal of the tonsils, and only a few cases have been reported. Pearlman and Salinger reported a fatal case coming under their observation and reviewed the literature on the subject. There seems to be no definite agreement as to the etiology of the symptoms of pallor, hyperpyrexia and occasionally death following anesthesia and operation. It would seem unwise, however, to operate on any child with a preoperative temperature above 99.2 F.; one should avoid general anesthesia if possible, and reduce operative manipulations to a minimum.

TREATMENT

Robey and Finland presented evidence that tonsillectomy may be performed during the active stage of acute rheumatic fever without harm to the patient from the operation. Tonsillectomy at that time offers no more dangers than when it is performed under what appear to be the most favorable conditions. Ether was the anesthetic selected for the majority of the 165 cases of this series. The only complication that delayed operation was an accompanying bronchitis or bronchopneumonia. Cardiac conditions practically never contraindicate operation except in extreme cases. The decision to operate was made by internists and not by rhinologists. In several cases in which symptoms have repeatedly recurred, the results of operation have been brilliant.

Levinger performed tonsillectomy on 235 patients with peritonsillar abscesses. In 60 of the patients the operation was immediately preceded by tonsillitis. On the basis of his experiences he came to the conclusion that the opinion now generally held, that tonsillectomy should not be performed during or shortly after an attack of acute tonsillar disease, is no longer tenable, because this expectant attitude does not always prevent complications. The author thought that complications were usually caused by too radical an operation at the lower pole of the tonsil. That complications did not develop during his tonsillectomies, in spite of the fact that acute inflammations existed, was considered to be the result of his procedure to avoid a complete removal at the lower pole. It was asserted that, provided all the deeper recesses are removed, a tonsillar stump may remain at the lower pole, because even radical removal does not prevent the aftergrowth of tonsillar tissue. The author stressed the fact that tonsillectomy not only is permissible during

the existence of a peritonsillar abscess but is the most favorable form of treatment, especially in cases of recurrent abscesses.

In a report on electrosurgical extirpation of the tonsils, Silvers described the various methods employed with their end-results. He concluded that the electrothermic technic has a definite place in otolaryngology. He described the advantages and disadvantages of the operative procedure.

Boutarel discussed the indications for and the methods of surgical diathermy. He stated that he preferred the spherical to the needle electrode as being less drastic in its effect and more easily visible. At the same time, he favored a continuous current. Complications are rare and are usually due to coagulation of an aberrant vessel. A secondary hemorrhage may result after loss of the eschar, but with the cautious use of the cautery such accidents can be prevented.

McKenzie expressed the conviction that diathermic coagulation will come to be the method of choice for removing tonsils in adults. It is unsuitable for children.

Schulte enumerated the following conditions in which roentgen treatment of the tonsils is indicated: (1) If anesthesia or operation is contraindicated; (2) if a secondary hemorrhage is to be feared because of arteriosclerosis; (3) if the tonsils are infected, with the result that an operation might disseminate the bacteria and cause a pulmonary abscess, septicemia or endocarditis; (4) if the regional lymph nodes are infected; (5) if the patient has a chronic cardiac disturbance, diabetes, exophthalmic goiter, chorea, rheumatism, hemophilia, asthma, tuberculosis, status lymphaticus or any disease that greatly lowers the bodily resistance; (6) if the patient has frequent attacks of peritonsillar abscess; (7) if the patient is a singer or a public speaker and has frequent attacks of sore throat and catarrh, and (8) if frequent attacks of sore throat and catarrh occur following tonsillectomy.

During the past three years Schönfeld and Baumbach have treated 150 children, aged from 3 to 13 years, with enlarged tonsils by means of roentgen irradiation. Of 106 of these children who were kept under observation the tonsils returned to their normal size in 41, became much smaller in 19 and remained unchanged in 40. Examination from two to three years after the completion of the treatment showed that in only 43 cases (41 per cent) were the tonsils smaller than they had been before irradiation. In all the patients, however, in whom the size of the tonsils had decreased following the treatment, breathing through the mouth during sleep had ceased, and in the great majority the previously frequent attacks of pharyngitis had disappeared completely. In none of the cases was the treatment followed by chronic injury of the mucosa or a disturbance in the functioning of the glands of the mouth and pharynx. On the basis of their experience in these cases the

authors expressed the belief that roentgen irradiation of enlarged tonsils is indicated particularly in patients with a history of frequently recurring infections of the pharynx.

According to Scal the treatment of inoperable tonsils with the implantation of removable platinum radon seeds is an adequate substitute for tonsillectomy in those cases in which surgical intervention is contraindicated. Only one treatment is necessary. There is no hospitalization and no anesthetic, and the patient does not have pain or discomfort in attending to his usual occupation. As compared with roentgen treatment, radium exerts its action only on the lymphoid tissues in the area implanted, without affecting the adjacent tissues. The roentgen rays affect all the tissues and result in annoying aftereffects. Electrocoagulation likewise can be used only by skilled hands; even then, there is the possibility of damaging the adjacent structures.

The advantages of suction tonsillectomy described by Waring are as follows: The technic is simple and easily mastered. It is applicable to all types of tonsils and can be used under local or general anesthesia. The operation is performed rapidly, therefore requiring only a short period of time under general anesthesia. The operation is comparatively bloodless, and all infected material from the tonsil is readily removed, thus minimizing the danger of aspiration into the lungs.

The use of the Waring method of suction tonsillectomy was reported by Jacobs and Trauner who reviewed their technic and results in a group of 389 cases. They concluded that tonsillectomy by the suction method is comparatively simple, but the technic must be acquired by practice. The entire tonsil and its capsule are removed without damage to the surrounding tissue or with a minimum of trauma. The operation is usually bloodless, and all infectious material is completely removed by suction so that the danger of postoperative pulmonary abscess or pneumonia is eliminated.

During the past ten years Stout has used the eversion method of tonsillectomy. The tonsil is grasped by a tenaculum forceps and drawn well out of its fossa. The Allis dissector or the Lott evertor is used to tear through the plica covering the lower portion of the tonsil. This allows the tonsil to evert more easily. The evertor is plunged into the upper part of the tonsil and the tonsil is everted. The operator than takes a second grasp including the upper and lower poles. A Lewis snare is applied, the loop is pressed against the pillars and the snare is tightened slowly. Stout and his colleagues have performed more than 15,000 operations by this method.

Bird related his experiences with the closed ethyl chloride and oxygen narcosis method in more than 300 tonsillectomies. He commented on its safety and freedom from postanesthetic complications.

In a study of the end-results of tonsillectomy with special reference to sinus infection in adults, Pond reported on a group of 108 cases. In his opinion the tonsils may become infected from the material which drains from the sinuses into the pharynx. In the group of 108 cases 5 patients were considered as cured. There were 3 cases of ethmoiditis and 2 of spheno-ethmoiditis. In 51 cases of various types there was definite improvement, and in 52 cases there was no benefit from tonsillectomy. The basis for cure or improvement of the sinusitis is based on the improvement of the general condition of the patient following tonsillectomy.

A new instrument for the control of tonsillar hemorrhage has been described by Alper. The instrument is composed of two round perforated knobs which end at their proximal extremity in a collar-like ridge containing a rubber ring. Pressure is maintained by a spring arrangement which not only keeps the pressure equal and constant, but which enables the patient to swallow while the instrument is in place.

Shewman described an instrument to control hemorrhage after tonsillectomy, which consists of a special forceps for inserting a skin clip into the tonsillar fossa:

A new tonsil forceps designed by Berger is adaptable to all types of tonsils, and enables one to grasp small tonsillar tags.

A new instrument for grasping tonsil tabs was described by Scal. The grasping end consists of two cuplike blades, which are serrated at their margins and fit accurately into each other.

A sponge-holder for applying pressure in the adenoid area has been devised by Schwartz.

BIBLIOGRAPHY

GENERAL CONSIDERATIONS

Bradley, W. H.: Tonsils and Nasopharyngeal Epidemics, Arch. Dis. Childhood **5**:335, 1930.

Hofer, I.: Relation Between Tonsils and Tracheobronchial Lymph-Nodes, Monatschr. f. Ohrenh. **64**:50, 1930.

Jerwood, B. E.: Tonsils, Teeth and Maternity, Brit. M. J. **2**:1196, 1929.

Kaiser, A. D.: Results of Tonsillectomy: A Comparative Study of Twenty-Two Hundred Tonsillectomized Children with an Equal Number of Controls Three and Ten Years After Operation, J. A. M. A. **95**:837 (Sept. 20) 1930.

McLean, C. C.: The Incidence of Recurrent Respiratory Infections in Childhood, J. A. M. A. **95**:1338 (Nov. 1) 1930.

Miller, M. V.: The Lingual Tonsil, Laryngoscope **40**:117, 1930.

Monroe, J. D., and Volk, V. K.: Effects of 736 Tonsillectomies and 741 Controls, Am. J. Pub. Health **20**:495, 1930.

Shambaugh, G. E.; Hayden, D. B.; Hagens, E. W., and Watkins, R. W.: Statistical Studies of the Children in Public Schools for the Deaf: Additional Report of Committee, Division of Medical Sciences, National Research Council, Arch. Otolaryn. **12**:190 (Aug.) 1930.

Snoke, P. O.: Lympho-Epithelioma of the Tonsil: A Bilateral Case, Arch. Otolaryng. **11**:602 (May) 1930.

Yankauer, S.: Nasopharyngeal Abscess: A Report of One Hundred Fifty-Five Cases, Ann. Otol. Rhin. & Laryng. **39**:481, 1930.

ANATOMY AND PHYSIOLOGY

Heiberg, K. A.: Modern Aspect of Anatomy of Tonsils in Relation to Germinal Centers and Lymphocytes, Folia oto-laryng. **19**:209, 1930.

Stout, P. S.: The Relative Position of the Deeper Vessels of the Neck with the End of the Needle in Local Anesthesia Around the Faucial Tonsil, Laryngoscope **40**:539, 1930.

BACTERIOLOGY AND PATHOLOGY

Brandes, M.: Specific Toxins of Bacteria Isolated from Tonsils of Patients with Articular Rheumatism, Ztschr. f. Hals-, Nasen- u. Ohrenh. **26**:318, 1930.

Castellani, A.: Fetor Oris of Tonsillar Origin and Certain Bacilli Causing It, Lancet **1**:623, 1930.

Fungal Diseases of Tonsils (Tonsillomycoses), J. Trop. Med. **33**:165, 1930.

Cobe, H. M.: Incidence of Bacteria in 400 Tonsil Cultures, J. Infect. Dis. **46**:298 (April) 1930.

Fleming, A., and MacLean, I. H.: Occurrence of Influenza Bacilli in Mouths of Normal People, Brit. J. Exper. Path. **11**:127, 1930.

Guttman, M. R.: Determination of Pathogenic Tonsil: Preliminary Report of New Biologic Test, Illinois M. J. **57**:352, 1930.

Vialle; LeCocq, and Ronchèse: Mixed Chancre of the Tonsil, Arch. internat. de laryng. **9**:513, 1930.

COMPLICATIONS

Ballenger, H. C.: Appendicitis Following Tonsillectomy: Report of Two Cases, Arch. Otolaryng. **12**:67 (July) 1930.

Carmody, T. E.: Pulmonary Abscess Following Tonsillectomy: Laryngologic Aspect, Arch. Otolaryng. **11**:200 (Feb.) 1930.

Clerf, L. H.: Pulmonary Abscess Following Tonsillectomy: Bronchoscopic Considerations, Arch. Otolaryng. **11**:192 (Feb.) 1930.

Dixon, O. J., and Helwig, F. C.: Thrombophlebitis of the Internal Jugular Vein as a Complication of Tonsillitis, Ann. Otol., Rhin. & Laryng. **39**:1137, 1930.

Goerke, M.: Pyemia Following Tonsillectomy: Prophylaxis and Treatment, Ztschr. f. Laryng. Rhin. **19**:287, 1930.

Guttman, M. R.: Alleviation of Pain in Peritonsillar Abscess: A Report of a Method Involving Cocainization of the Palatine Nerves Passing Through the Sphenopalatine Ganglion, Arch. Otolaryng. **11**:426 (April) 1930.

Hara, H. J.: Aspiration in Tonsillectomy; Comparative Merits of Posture and Other Factors: Bronchoscopic Study of 110 Patients, California & West. Med. **33**:628, 1930.

Hartz, W.: Complications of Tonsillectomy, Laryngoscope **40**:598, 1930.

Hochfilzer, J. J.: Phlegmon of the Neck and Osteomyelitis of the Mandible Following Tonsillectomy, Arch. Otolaryng. **12**:175 (Aug.) 1930.

Holman, E.: Posttonsillectomic Pulmonary Abscess: Factors in Healing, Arch. Otolaryng. **11**:287 (March) 1930.

Pearlman, S. J., and Salinger, S.: Hyperpyrexia (Fatal) Following Tonsillectomy, Ann. Otol. Rhin. & Laryngol. **39**:502, 1930.

Pierson, P. H.: Posttonsillectomic Pulmonary Abscess: Medical Aspects, Arch. Otolaryng. **11**:279 (March) 1930.

Schall, L. A.: Pulmonary Abscess Following Tonsillectomy: Bronchoscopic Considerations as an Aid to the Surgeon, Arch. Otolaryng. **11**:300 (March) 1930.

Shapiro, S. L.: Deep Cervical Infection Following Tonsillectomy, Arch. Otolaryng. **11**:701 (June) 1930.

Stein, S.: Bacillus Proteus Vulgaris Infection in Secondary Mastoiditis, Following Tonsillectomy, and in Radium Implantation in Tonsils, Ann. Otol. Rhin. & Laryngol. **39**:1155, 1930.

von Hofe, F. H.: Emphysema of the Head and Neck Complicating Tonsillectomy, J. A. M. A. **95**:934 (Sept. 27) 1930.

Watson-Williams, E.: Parapharyngeal Abscess: Quinsy That Isn't, Lancet **1**: 792, 1930.

Weidlein, I. F.: Chronic Abscess of Pharyngomaxillary Fossa Without Symptoms: Report of a Case, Laryngoscope **40**:583, 1930.

White, J. W.: Infection Involving the Pharyngo-Maxillary Space: Report of Two Cases, Arch. Otolaryng. **12**:248 (Aug.) 1930.

TREATMENT

Alper, I. I.: A New Instrument for the Control of Tonsillar Hemorrhage, Arch. Otolaryng. **11**:86 (Jan.) 1930.

Berger, M.: New Tonsil Forceps, Arch. Otolaryng. **12**:376 (Sept.) 1930.

Bird, H. M.: Anesthesia for Short Operations on Children, Especially Tonsillectomy, Brit. M. J. **2**:147, 1930.

Boutarel, M.: Thermacoagulation of Tonsils, Paris méd. **2**:476, 1929.

Jacobs, S. N., and Trauner, L. M.: Suction Tonsillectomy: Waring Method, Laryngoscope **40**:444, 1930.

Levinger, S.: Should Tonsillectomy be Performed During and Shortly After an Attack of Tonsillitis? München. med. Wchnschr. **77**:1666, 1930.

McKenzie, D.: Diathermy Extirpation of Pharyngeal Tonsils, Brit. M. J. **2**:951, 1929.

Pond, C. W.: Some End-Results of Tonsillectomy, with Special Reference to Sinus Infection in Adults, Laryngoscope **40**:286, 1930.

Robey, W. H., and Finland, M.: Effect of Tonsillectomy on Acute Attack of Rheumatic Fever, Arch. Int. Med. **45**:772 (May) 1930.

Scal, J. C.: The Inoperable Tonsil: Treatment with Radium in Preference to Other Nonoperative Methods, Arch. Otolaryng. **11**:459 (April) 1930.

A New Instrument for Grasping Tonsil Tabs, Arch. Otolaryng. **11**:621 (May) 1930.

Schönfeld, H., and Baumbach, G.: Enlarged Tonsils: Roentgen Treatment, Strahlentherapie **36**:472, 1930.

Schulte, G.: Diseases of Tonsils: Roentgen Treatment, Strahlentherapie **36**:708, 1930.

Schwartz, L., Jr.: An Adenoid Sponge-Holder, Arch. Otolaryng. **12**:376, (Sept.) 1930.

Shewman, A. W.: Instrument to Control Hemorrhage After Tonsillectomy, Arch. Otolaryng. **12**:526 (Oct.) 1930.

Silvers, L. J.: Electrosurgical Extirpation of the Tonsils: A Clinical Study of the Various Methods Employed, With Their End-Results, Arch. Otolaryng. **12**:511 (Oct.) 1930.

Stout, P. S.: Tonsillectomy by the Eversion Method, Laryngoscope **40**:663, 1930.

Waring, J. B. H.: Suction Tonsillectomy, Arch. Otolaryng. **12**:31 (July) 1930.

News and Comment

The Colorado Congress of Ophthalmology and Oto-Laryngology.—The ninth annual summer graduate course and the Colorado Congress of Ophthalmology and Oto-Laryngology will be held from July 20 to August 1, inclusive. The class will be divided into small sections for the demonstrations and clinics. Daily complimentary luncheons followed by round table discussions will be continued as a popular feature. The Congress will be held on Friday and Saturday, July 24 and 25, for the presentation and discussion of special papers. At the completion of the program, on the afternoon of July 24, there will be a motor trip into the Rocky Mountains followed by a complimentary banquet at the Mountain Home of the Motor Club of Colorado. Among those who have consented to give special courses this year are: Dr. C. S. O'Brien, Iowa City; Dr. F. H. Adler, Philadelphia; Dr. H. J. Howard and Dr. French K. Hansel, St. Louis; Dr. A. C. Furstenberg, Ann Arbor, Mich., and Dr. W. J. McNally, Montreal, Canada. Applications for the graduate course, accompanied by a check for $50, the total fee, should be sent to Dr. H. L. Whitaker, corresponding secretary-treasurer, 1612 Tremont Place, Denver.

Post-Graduate Course at University of Bordeaux.—A post-graduate course in ear, nose and throat surgery will be given by Prof. George Portmann in English at the University of Bordeaux, France, beginning July 27, 1931. The class is limited to twelve physicians. Further information may be secured from Dr. Leon Felderman, 413 Mitten Building, Philadelphia.

Abstracts from Current Literature

Ear

THE USE OF INSULIN IN POSTOPERATIVE MASTOIDITIS WITH PROLONGED DRAINAGE. M. S. ERSNER and J. J. PRESSMAN, Ann. Otol. Rhin. & Laryng. **39**:1125 (Dec.) 1930.

Delayed healing of mastoid wounds is frequently encountered in patients whose general condition is below normal, even though there is no reason to believe that cells have been left unopened or that anything but complete evacuation and thorough drainage had been accomplished at the time of operation. Such patients usually have been acutely ill for a long time; they have become anemic, have poor appetites, and have developed a low resistance, all of which has a definite bearing on the progress of the mastoid wound. In order to improve the physical status of the patient, the authors have adopted the use of subcutaneous injections of insulin in cases of delayed healing and prolonged suppuration following mastoidectomy in nondiabetic persons. In a comparatively small series of cases the results have been very satisfactory. Repeated observations of many workers has revealed that in malnourished persons the use of insulin increases the appetite and brings about a gain in weight together with an improvement in general well being. Single daily doses of insulin were administered, the patients being instructed to report one-half hour before lunch for the injections. In children the usual initial dose was three units, being rapidly increased to five or more, according to the reaction observed and the ability of the patient to tolerate larger doses. A diet rich in fats rather than carbohydrates is logical, for the appetite created by the use of insulin is soon lost if carbohydrate is taken early in the meal. The treatments were usually started during the sixth or seventh week of convalescence. In each case, in keeping with the improvement in general health and appetite, there followed a rapid decrease in drainage, new granulations appeared, and rapid and complete healing occurred.

SNAPP, Grand Rapids, Mich.

TREATMENT OF THE DEAF INFANT. G. DE PARREL, Progrès méd., Dec. 13, 1930, p. 2208.

The author classifies deaf infants in three groups: deaf-mutes, partially deaf children and those hard of hearing. The deaf-mutes should be given over to educational specialists at 3 years of age. The partially deaf infants frequently show rhinolaryngologic defects and should be examined thoroughly, after which special education is indicated. The children who are hard of hearing should be in special classes of not over eight or ten pupils.

SANFORD, Chicago.

CLINICAL ASPECTS OF INFLAMMATORY DISEASES OF APEX OF PETROUS PORTION OF THE TEMPORAL BONE. E. GRABSCHEID, Monatschr. f. Ohrenh. **65**:289 (March) 1931.

Grabscheid states that the inflammatory diseases of the apex of the petrous portion of the temporal bone have received more attention in recent years. It was found that the symptoms on the part of the abducens, which were formerly considered characteristic, were less frequent than the symptoms from the trigeminus. In regard to the anatomic foundations of the diseases of the apex of the petrous part, opinions are still divided. Some observers consider extradural or osteitic foci the cause, whereas others assume a local meningitis. Recent investigations by Brummer have proved that in the majority of cases a serous meningitis is the cause. The author describes two cases. In both patients there existed an acute otitis together with a completely developed mastoiditis, and following the opera-

tion serous meningitis developed. As sign of the serous meningitis, there existed in the first case a sensitiveness to pressure and to percussion in the malar process, also dermographism, stiffness of the neck, high temperature, Kernig's sign, slight stupor, positive Nonne-Apelt reaction and postoperative increase in temperature. In the second case there were high temperatures, dermographism and positive Nonne-Apelt and Pandy reactions in the cerebrospinal fluid. The clinical manifestations of serous meningitis are not characteristic. However, the term serous meningitis is significant for differentiation from the suppurative form, and there is nearly always a complete recovery after serous meningitis. The two patients observed by the author also recovered. The increased pressure of the cerebrospinal fluid, which some consider as a significant symptom of serous meningitis, the author considers of minor importance, because it is influenced by too many factors, so that it is difficult to interpret slight fluctuations. However, he attaches greater importance to an increase of the albumin and of the globulin. Of the symptoms indicating an irritation of the trigeminus, which are frequently manifest in diseases of the apex of the petrous portion of the temporal bone, the author mentions pains in the temple and in the depth of the orbit, pounding pain in the depth of the ear and toothache. However, the most characteristic symptom of a meningitis localized on the apex of the petrous part is the hyporeflexia of the cornea on the side of the diseased ear.

EDITOR'S ABSTRACT.

PATHOGENESIS AND CLINICAL ASPECTS OF TRAUMATIC INJURIES OF THE INTERNAL EAR. M. GOERKE, Ztschr. f. Rhin., Laryng. (teil 1: Folia otolaryng.) **20**:363 (March) 1931.

Goerke differentiates between direct and indirect injuries of the internal ear according to the manner of attack of the force which causes the trauma. Aside from gunshot injuries during the war, direct injuries of the internal ear are rare. Of the indirect injuries those that occur in fractures of the base of the skull are discussed first. Especially dangerous for the internal ear are transverse fractures. The peculiar brittleness of the petrous portion of the temporal bone is the cause of the development of small fissures in the labyrinth even when the line of fracture does not involve the aural region. Recent observations have proved that such fissures are much more frequent than is generally believed. However, in many cases they are so small that only microscopic examination will reveal them. These fissures may sometimes be the starting point of a late meningitis. The interval between the formation of the fissures and the manifestation of the meningitis may be of considerable length (the longest interval known is 210 days). In cases of otogenetic meningitis, in which the anamnesis reveals a cranial trauma even several months before the meningitis, a causal relationship cannot be denied. Fissures may also cause an ossifying periosteitis, which may lead to an obliteration of the labyrinthine spaces and to increasing deafness. Another group of lesions of the internal ear are those designated as commotio labyrinthi; they are caused by a dull blow on the head or by a fall. In these cases the relation between the trauma and the injury of the internal ear is more difficult to recognize than in the traumas previously mentioned. In regard to the nature of the changes in commotio labyrinthi, it is stated that the former theories, which assumed intralabyrinthine hemorrhages, distortions, misplacements and tearing of the soft parts of the labyrinth, have been largely disproved and that more recent investigations have revealed that the changes in commotio labyrinthi are, on the one hand, vascular changes such as marked distention of the vessels, diapedesis of the leukocytes and their perivascular accumulation and, on the other hand, pronounced degenerative changes in the cells of the nuclei of the acoustic nerve. The author further discusses labyrinthine lesions, in which although the cranium is not directly affected by the trauma, the influence on the auditory organ cannot be denied. In the majority of these cases the injury of the internal ear is produced by less severe but by continuously acting influences. Nevertheless, there are also cases on record in which one single loud tone or noise has caused an auditory disturbance.

Investigations have revealed that the injuries caused by impacts of air are due to changes in the nervous apparatus, but opinions still differ as to how these changes develop. Some observations revealed that if the physiologic conduction of sound was interrupted, injuries in the internal ear did not develop. This would justify closure of the ears with cotton or wax in workers obliged to work in noisy surroundings. However, others observed that among soldiers who operated heavy artillery guns, and who had closed their ears with cotton, auditory disturbances were more frequent than among those who had not taken these protective measures. The author thinks that a blast may reach the ear by three routes: the aerotympanal the osteotympanal and the ossal. If the influence is only acoustic the physiologic route is taken; however, in case of a concussion the ossal route is used. The author thinks that the fact that some ears are injured by noise more easily than are others is due to the condition of the ear. A diseased ear is more likely to be injured by a noise than a healthy ear, and it is also probable that constitutional factors create a predisposition. Of course, even healthy ears may be injured by continued noises, but the changes that develop are generally reversible, provided that periods of rest are introduced which make it possible for the ear to recover from the strain.

EDITOR'S ABSTRACT.

ROENTGEN DIAGNOSIS IN MASTOIDITIS. K. SCHEUER, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **20**:437 (March) 1931.

According to Scheuer, roentgenoscopy of the mastoid process is still new. He therefore considers it advisable that experiences should be reported and errors pointed out. As yet the roentgen method is imperfect, so that it can be only an auxiliary method. Correct interpretation of the roentgenograms is especially difficult. After describing the technic of roentgenoscopy that is employed in the university clinic in Würzburg, the author compares the results of roentgenoscopy with the operative findings in 104 cases of mastoiditis. He found that the roentgenogram had been interpreted correctly in 78 and incorrectly in 25 cases. In the incorrectly diagnosed cases, in 11 in which the roentgenograms were negative the operative findings were positive and in 14 in which the roentgenograms were positive the operative findings were negative. These statistics show that the roentgenogram may simulate disintegrating processes that in reality do not exist. In analyzing these cases it was found that the causative factor in most incorrect diagnoses is the sinus canal. Deception is produced by the fact that the sinus canal becomes visible in a certain circumscribed portion; the lessened density may be interpreted as indicative of a disintegrated area. The sinus always becomes visible when the air content of the cells covering it has decreased to such an extent that its shadow is no longer covered. The decreased air content may be caused by the following conditions: 1. The air in the cells may have been displaced by exudate. 2. The sinus may no longer be covered by cells, (many operations have corroborated this) so that the conditions are the same as in a compact mastoid process. In the latter condition likewise the sinus becomes visible; the air-containing cells which cover its shadow are absent. In all except four of these cases there were mastoid processes with inhibited pneumatization, so that the roentgen pictures, even when the operative results were known, gave no sure indications of changes in the bone. In such mastoid processes the stereoscopic method of exposure has been found helpful. The results of this method will be reported later after a sufficiently large amount of material has been studied.

EDITOR'S ABSTRACT.

HISTOLOGIC OBSERVATIONS ON FENESTRAE IN ACQUIRED DEAFNESS AND COMMENT ON HEALING OF SURGICAL INJURY OF THE STAPES. H. BRUNNER, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:79 (March 3) 1931.

Brunner reports the case of a woman, aged 25. After a short report of the clinical history, he gives a detailed description of the results of necropsy. This

case is significant because it shows: (1) the changes in the internal ear in deafness following meningitis, (2) the changes in the mucous membrane of the middle ear many years after a radical operation and (3) the microscopic picture of the operative injury of the stapes with its sequelae. The results of the microscopic examination of the right temporal bone are summed up as follows: (1) suppurative infiltration of the mucous membrane of the tube and of the middle ear; (2) penetration of the cylindric epithelium into the upper wall of the external auditory meatus and of the epidermis into the middle ear; (3) a healed osteitic focus in the upper wall of the bony auditory meatus; (4) luxation of the stapes against the middle ear; (5) partial closure of the fenestra ovalis by a membrane of connective tissue; (6) a healed osteitic focus on the upper and the lower frame of the fenestra ovalis; (7) obliteration of the fenestra rotunda by connective tissue; (8) a connective tissue canal in the lamina spiralis ossea secundaria; (9) partial ectasis and partial collapse of the membranous canal of the cochlea; (10) connective tissue strands in the scala tympani; (11) atrophy of the organ of Corti; (12) marked atrophy of the nerve ganglion apparatus of the cochlea; (13) a vestibulocochlear septum, and (14) dilatation of the ductus endolymphaticus. The following conditions were found on the left side: (1) suppurative infiltration of the mucous membrane of the tube and of the middle ear; (2) a bony canal above the fenestra ovalis; (3) luxation of the stapes against the middle ear; (4) a fissure of the base of the stapes; (5) a bony canal underneath the fenestra rotunda; (6) atrophy of the spiral nerve and the spiral ganglion; (7) atrophy of the trunk of the cochlear nerve; (8) a vestibulocochlear septum; (9) dilatation of the endolymphatic and utriculosaccular ducts; (10) adhesions between the base of the stapes and the free wall of the utriculus, and (11) an increased number of the cartilage-containing, interglobular spaces.

EDITOR'S ABSTRACT.

METHOD TO PROVE ABSENCE OF OBTURATING SINUS THROMBOSIS OR JUGULARIS THROMBOSIS IN A CASE IN WHICH THERE WAS A PYEMIC FEVER CURVE. O. MUCK, Ztschr. f. Hals-, Nasen- u. Ohrenh. **28**:98 (March 3) 1931.

Muck points out that it is sometimes difficult to decide whether an obturating thrombosis exists in the sinus transversus or in the sinus jugularis. In doubtful cases in which it is difficult to decide whether the sinus should be opened or the jugularis should be ligated, he employs a special diagnostic method. As compression of the jugular vein of one side produces a venous murmur on the other side or intensifies the already existing murmur, especially in children, the author employed this method when a pyemic fever curve existed in the course of an acute purulent otitis media. The positive outcome of this test indicates that an obturating thrombosis does not exist. The author stresses the fact that in order to avoid unnecessary and dangerous operations on the sinus, this method should be employed only in doubtful cases. However, if sinus phlebitis is evident, this differential diagnostic aid should be omitted, and the necessary therapeutic measures should be carried out as soon as possible.

EDITOR'S ABSTRACT.

THE SEDIMENTATION OF ERYTHROCYTES IN INFECTIONS OF THE MIDDLE EAR. V. HLAVÁČEK, Časop. lék. česk. **67**:1395 (Oct. 5) 1928.

Cases of mastoiditis showed increased sedimentation of red cells independent of temperature. Extensive lesions were found in all cases of accelerated sedimentation. The figures for increased sedimentation were comparable with those for extensive pathologic processes, such as tuberculosis, with marked elevation of temperature. Practically only the decreased sedimentation time is of value with other signs and symptoms in deciding as to operation. In cases of decreased sedimentation no tissue detritus except nonpurulent cholesteatoma was noted.

STULIK, Chicago.

STATISTICAL REPORT ON THE CAUSE OF DEAF-MUTISM IN THE VYMOLA INSTITUTE IN PRAGUE. VÝMOLA, Časop. lék. česk. **67**:1525 (Nov. 2) 1928.

In 112 patients, the diagnosis on objective indications was congenital deaf-mutism in 71, acquired deaf-mutism in 37 and deaf-mutism of undetermined origin in 4. On careful checking of the family history it was found that the condition was truly congenital in only 53. The reasons cited for acquired deaf-mutism were: undetermined fever, 2 cases; pertussis, 1 case; grip, 2 cases; meningitis, 1 case; otitis media, 1 case; measles, 1 case; convulsions, 3 cases; trauma (fall), 4 cases; fright, 2 cases, and difficult teething, 1 case. The causes given in the series cited are: consanguinity, mendelian heredity, mongolism, constitutional diseases, alcoholism, infectious diseases and social environment.

STULIK, Chicago.

Pharynx

OPERATIVE PROCEDURE FOR MAKING PASSABLE THE CICATRICALLY OCCLUDED NASOPHARYNX. Z. WEIN, Monatschr. f. Ohrenh. **65**: 352 (March) 1931.

Wein discusses that group of obstacles to nasal breathing that are caused partly by cicatricial occlusion of the portion of the nasopharynx that is posterior to the choanae (retronasal synechias and atresias) and partly by cicatricial adhesions of the soft palate to the posterior wall of the pharynx (palatopharyngeal synechias). As causes of these conditions, he mentions syphilis, leprosy, lupus, diphtheria and faulty technic in tonsillectomy. In discussing the disadvantages of these occlusions, he points out that the hinderance to nasal respiration has a detrimental influence on the respiratory passages, on the elasticity of the lungs and on the circulation. The stasis of the nasal secretion is not only unpleasant but it also leads to catarrh of the nasal cavity and frequently to sinusitis. The sense of smell is entirely absent; the taste is dulled, and the absence of nasal breathing has also a detrimental effect on the auditory organ. The author further reviews the various methods that have been employed for the treatment of the occluded nasopharynx, and then describes his own operative procedure which he used on a woman, aged 50. Following cocainization of the nasal and of the pharyngo-oral cavities, he anesthetizes the outer part of the nose, the nasal septum and the cicatricial tissues in the nasopharynx by injecting a 1 per cent solution of procaine hydrochloride. He then makes the incisions according to Schloffer's method so that the outer portion of the nose can be entirely turned aside. Following bilateral separation of the mucous membrane from the nasal septum, he performs a submucous resection of the portion of the septum that is in the nasal cavity proper. The cartilage and the bone of the septum is removed to such an extent that on the base of the skull as well as on the base of the nose there remain only ridges of bone from 2 to 3 mm. high. Then the cicatricial tissues are removed, and following this, the soft palate is lifted from the posterior wall of the pharynx. Two pedicle flaps, obtained from the mucous membrane coverings of the nasal septum, are used to cover the surfaces of the wound. The mucous membrane grafts are fixed by means of an iodoform gauze tamponade, which is left in place for one week. The author further describes the after-treatment for the purpose of dilatation, and the results of this surgical treatment. There is no longer stasis of the nasal secretion. The nasal respiration is sufficient; the sense of smell gradually returns, the pharynx is no longer dry, and the soft palate has almost entirely regained its elasticity. The patient feels much better than before the operation. The author thinks that in severe cases of retronasal and palatopharyngeal adhesions his method brings better results than those formerly employed. Of course, Schloffer's method, which permits a turning aside of the outer portion of the nose, is advisable only in cases in which there are extensive adhesions. However, the resulting cosmetic defect is of minor significance when it is considered that such an operation is generally necessary in persons in whom the nose is already deformed.

EDITOR'S ABSTRACT.

ANGINA AGRANULOCYTOTICA. BAYER, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **20**:375 (March) 1931.

Bayer reports the clinical history of a woman aged 51. Following extraction of several suppurating roots of teeth she was troubled with severe pains in the gums and with headaches. Later she had chills, sore throat, headaches and slight fever. The physician in charge diagnosed the case as tonsillitis. Shortly after that the patient again had fever and inflammation of the throat and the tongue. A peritonsillar abscess was discovered and was incised. Later, necrotic processes developed on various portions of the oral mucous membrane and on the genitalia. The blood picture revealed the characteristics of agranulocytosis, and treatment with arsphenamine was then resorted to. The patient was given an intravenous injection of 0.45 Gm. of neoarsphenamine and simultaneously an intramuscular injection of 0.3 Gm. of myoarsphenamine. The effect of this large dose of arsphenamine was surprising. The patient no longer had difficulty in swallowing, the necrotic processes healed gradually and the blood picture improved. In a comparatively short time the patient recovered completely and was discharged from the hospital. Six months later, however, the patient had a relapse, and this time neoarsphenamine was without effect. The patient died. In conclusion it is pointed out that it is not entirely new to employ arsphenamine in cases of this type, but that in the majority of cases it fails, probably because of insufficient doses. The good results in the reported case are ascribed to the large dose, and the author advises that in such cases the maximum doses should be given at the first administration in order to overcome the organism's biologic lethargy.

EDITOR'S ABSTRACT.

ARREST OF HEMORRHAGE AFTER TONSILLECTOMY: A REPLY TO AN ARTICLE BY GUMPERZ. E. KRONENBERG, Ztschr. f. Laryng., Rhin. (teil 1: oto-laryng.) **20**:458 (March) 1931.

Kronenberg replies to an article by Gumperz, in which the latter emphasized the necessity of the complete removal of the tonsil and the ligation of all vessels. Kronenberg shows that Gumperz' emphasis on these points is superfluous, since these principles are known and adhered to by every competent surgeon. Kronenberg also takes exception to Gumperz' statements that chemical hemostatics may be employed to produce escharotic effects, and that they may be used as substitutes for the careful ligation of the vessels.

EDITOR'S ABSTRACT.

Nose

CORK AS PLASTIC MATERIAL FOR THE CORRECTION OF SADDLE NOSES. H. DAHMANN, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **20**:451 (March) 1931.

Dahmann first gives a general review of the various plastic methods. He discusses (1) transplantation of flaps of skin and connective tissue and (2) subcutaneous implantation of supporting materials. In regard to the first method he says that the cosmetic results are rarely satisfactory. In discussing subcutaneous implantation he first evaluates autoplastic materials, such as rib cartilage, iliac crest or a piece of the tibia. Preparatory to evaluating the various alloplastic materials he points out that alloplasty is contraindicated whenever extended cicatricial adhesions exist between the skin of the nasal bridge and the underlying bone. In such cases the skin, when lifted, is too thin and does not contain a sufficient number of vessels to guarantee adequate nutrition. In these cases living tissues, that is, autoplastic materials are required that form organic connections. Of the various alloplastic materials the author mentions paraffin, ivory and cork. The use of paraffin has been almost completely abandoned on account of its many disadvantages. Ivory, as plastic material, brings more favorable results than

paraffin; nevertheless, it also has certain disadvantages. A slight blow on the nose causes intense pain or may lead to the development of a hematoma, the resorption of which is extremely slow. Another disadvantage is the heavy feeling and the sensation of coldness during the cold season. The use of cork as plastic material has many advantages. It is resistant and is not subject to resorption. At any rate, if there is resorption it must be slight, for in patients in whom the cork was implanted more than four years ago, the condition is not yet changed. Other advantages of cork are that it is soft, elastic, light and a poor conductor of heat. In describing the technic the author emphasizes the advisability of selecting cork that has only a few pores and not too many stone cells. The preparation of the cork for the implantation requires five days. At first the piece of cork is cut comparatively large. It is boiled for one hour and then dried. On the following day it is cut a little smaller and boiled and dried again. On the third day it is subjected to the same process. On the fourth day it is given the shape required for the implantation, boiled again, then placed (from five to ten minutes) into a boiling solution of potassium permanganate (1:1,000), rinsed in boiling water, discolored in a hot 5 per cent solution of sodium bisulphite, washed again in boiling water and then dried. On the fifth day, the day of the operation, the implant is boiled for ten minutes, placed in a physiologic solution of sodium chloride and then implanted. The incision for the implantation is made in the groove above the left ala nasi. The pocket, which is to receive the implant, should be large enough so that the cork can be introduced without creating tension.

EDITOR'S ABSTRACT.

Miscellaneous

PRACTICAL SIGNIFICANCE OF CORRECT POSITION OF PATIENT IN OTOSURGICAL INTERVENTIONS IN ORDER TO AVOID SECONDARY INJURIES. G. ALEXANDER, Monatschr. f. Ohrenh. **65**:277 (March) 1931.

Alexander points out that in otosurgical interventions the correct position of the patient, that is, the topographic adjustment of the head, is of vital importance. The approach to the otosurgical regions is always from the antrum. The antrotomy is the technical foundation of even the most remote otosurgical interventions on the brain. The otosurgical exposure follows rules that are based on the topographic arrangement of the different parts. These geometric relations are guilty only when the head is in a certain position. A slight change in the position of the head causes considerable changes in the topography of the surgical field so that such terms as vertical, to the front, upward and backward can no longer be followed. It is therefore essential that before the surgeon commences to operate he sees that the patient's head is in the proper position. Of the surgical interventions in which the position of the head is of vital importance, the author mentions particularly paracentesis, antrotomy, removal of the mastoid process without exposure of sinus or dura and exclusion of the jugularis. He shows what complications may arise if the head is not in the correct position and gives diagrams showing the correct and the incorrect positions.

EDITOR'S ABSTRACT.

Society Transactions

AMERICAN SOCIETY FOR THE STUDY OF DISORDERS OF SPEECH

ROBERT WEST, PH.D., *Editor*

Chicago, Dec. 30 and 31, 1930, and Jan. 1, 1931

ELMER L. KENYON, M.D., *Chairman*

A SYMPOSIUM ON STUTTERING (STAMMERING)

THE PHENOMENOLOGY OF STUTTERING. DR. ROBERT WEST, Madison, Wis.

THE EDUCATIONAL APPROACH TO THE PROBLEM OF STUTTERING. DR. H. J. HELTMAN, Syracuse, N. Y.

CAN CLINICAL PROCEDURE IN THE TREATMENT FOR STUTTERING BE USED IN THE PUBLIC SCHOOLS? MISS PAULINE B. CAMP, Madison, Wis.

HOW DR. G. HUDSON MAGUEN TREATED STAMMERING. MISS MARY SUMMERS STEEL, Philadelphia.

STAMMERING AS AN IMPEDIMENT. DR. C. S. BLUEMEL, Denver.

CORRECTION OF STAMMERING IN BIG SCHOOL SYSTEMS. MISS LETITIA RAUBICHECK, New York.

BASIS FOR WORK WITH STUTTERERS IN GRAND RAPIDS PUBLIC SCHOOLS. MISS EUDORA P. ESTABROOK, Grand Rapids, Mich.

TWELVE YEARS OF VISUAL TREATMENT FOR STUTTERING. MISS M. CLAUDIA WILLIAMS, Cleveland.

METHODS USED IN THE TREATMENT OF THE STUTTERING CHILD IN THE PUBLIC SCHOOLS OF OMAHA (NEBRASKA). MISS ALICE LILJEGREN, Omaha.

RELATIONSHIP OF PERSONALITY AND BEHAVIOR DIFFICULTIES TO DISORDERS OF SPEECH. DR. PAUL L. SCHROEDER and MR. LUTON ACKERSON, Chicago.

BREATH CONTROL IN STAMMERING. MR. SAMUEL D. ROBBINS, Boston.

DUNLAP'S THEORY OF THE TREATMENT FOR STUTTERING. DR. JOHN M. FLETCHER, New Orleans.

STUTTERING. DR. SMILEY BLANTON, Poughkeepsie, N. Y.

A CONSIDERATION OF SOME OF THE PSYCHOLOGIC CAUSES AND TREATMENT OF STAMMERING. MRS MABEL FARRINGTON GIFFORD, San Francisco.

WHY VISUALIZATION IS THE BEST METHOD FOR STAMMERING. DR. WALTER B. SWIFT, Boston.

THE CORRECTION OF STAMMERING IN DETROIT. MISS CLARA B. STODDARD, Detroit.

THE TREATMENT FOR STUTTERING IN THE PUBLIC SCHOOLS OF WISCONSIN. MISS LAVILLA A. WARD, Madison, Wis.

SOME INTERPRETATIONS OF RECENT RESEARCHES IN THE CORRECTION OF STAMMERING. DR. ELIZABETH A. MCDOWELL, New York.

THE READJUSTMENT OF THE STUTTERER'S SPEECH. MISS JENNIE HEDRICK, Washington, D. C.

DIAGNOSIS AND TREATMENT IN CASES OF STUTTERING. DR. LEE EDWARD TRAVIS, Iowa City.

Oral Stammering: One of the Manifestations of the Conflicts of the Organic Stammerer. Dr. Charles G. Stivers, Los Angeles.

Speech Training and Mental Hygiene Method for the Correction of Stammering. Miss Mildred A. McGinnis, St. Louis.

The Problem of Stuttering. Mr. Frederick W. Brown, New York.

The Nature and Analytical Treatment of Stammering. Dr. Isador H. Coriat, Boston.

Treatment for Stuttering. Mr. Bryng Bryngelson, Minneapolis.

Stuttering. What About It? Dr. James Sonnett Greene, New York.

Conscious Detailed Psychomuscular Control of Speech Production as an Effective (if Not Necessary) Basis for All Manner of Psychologic Treatment for Stammering. Dr. Elmer L. Kenyon, Chicago.

Neuropedagogical Process of Treating Stammerers and Stutterers at Ohio State University. Dr. G. Oscar Russell, Columbus, O.

Although limitation of space prevents a detailed abstracting of these papers, certain interesting observations can be made on the program as a whole. A mere statement of the positions assumed by various speakers on a few of the outstanding points of difference will reveal the theories held by these authorities perhaps better than lengthy quotation from their papers.

Is Stuttering a Neurotic or Psychoneurotic Manifestation?

For:

Dr. Smiley Blanton, Poughkeepsie, N. Y.: Stuttering is a symptom of the inability of a child to adjust himself to the group. It is caused by fear, timidity or a negative (hate) attitude toward the group. The child wishes to talk with his conscious mind, but with the unconscious mind there is an inhibition against speech. Stuttering is the result.

Psychoanalysis has made a valuable contribution to the cause and treatment of stuttering. The first stage of the love life of the child is the oral erotic stage in which the child not only uses the mouth to get nourishment, but also to get sexual pleasure of the infantile type. The region of the mouth is highly charged with emotion, and sounds are made partly because of the pleasure they give through the stimulation of the vocal organs. This oral erotic stage passes normally. Sometimes, however, the child remains fixed in this stage, his love energy is centered on himself, and he becomes narcissistic. It is this narcissism that gives rise to timidity and self-consciousness.

Dr. Isador H. Coriat, Boston: Stammering becomes a neurosis in which the fixation of the libido at the developmental stage of oral erotism persists into maturity. In his unconscious, and likewise in the conscious, motor speech reactions, the stammerer remains fixed or anchored to this primitive biologic stage because he has been unable to sublimate, or has been able to sublimate only to a very limited degree, the original oral pleasure.

Mr. Samuel D. Robbins, Boston: I regard stammering primarily as a mental ailment, varying in intensity with the degree of the emotion the stammerer happens to be experiencing.

Mrs. Mabel Farrington Gifford, San Francisco: It must be fully understood in the beginning that according to my theory the causes of these nervous disorders of speech are psychologic, and that the spasmodic manifestation of the speech organs is only the external symptom of the deepseated mental conflict. It has now been definitely established that severe shocks and emotional conflicts in very early childhood remain as subconscious memories for many years and may continue to disturb the function of speech, which in itself is perfect, until corrective measures are applied. One can understand more easily the relation between stuttering and the subconscious emotional memories and conflicts when one considers that every normal speech reflects the momentary emotional state of mind.

DR. CHARLES G. STIVERS, Los Angeles: Stammering, in common usage, is recognized as an oral manifestation of emotional instability. Verbal stammering, however, is but one manifestation of the deeper and broader emotional maladjustment that finds an outlet in deviation from a normal or organic rhythm of life.

MR. FREDERICK W. BROWN, New York: The cause of this maladjustment is, I believe, emotional, and stuttering is but a symptom of the lack of integration of the personality of the stutterer.

DR. JAMES SONNETT GREENE, New York: Just how to apportion heredity and environment as etiologic factors in stuttering is about as contentious as in any other field. From all indications, stuttering is primarily psychologic rather than biologic. By it being psychologic, I mean that the stutterer is influenced by factors of training — the likes and dislikes, the bents, slants and attitudes, the character that one as an individual brings to one's daily actions and associations. The stutterer cannot directly inherit stuttering speech, but without doubt he inherits his neuropathic constitution, and if he lives in tense surroundings, almost any disturbance is liable to start the stuttering. Most histories in cases of stuttering show nervous traits in the patient, in the parents and in other members of the family.

Against:

DR. LEE EDWARD TRAVIS, Iowa City: It is my opinion that few stutterers need a searching psychoanalysis any more than most so-called normal persons. I am not aware of any experimental or clinical evidence that stuttering is a manifestation of an anxiety neurosis or anxiety hysteria. If the stutterer presents a morbid anxiety, it is mainly an effect and not a primary cause. As I have repeatedly maintained, the anxiety and fears of the stutterer are reactions to, and are developed after, the appearance of his defect.

MR. BRYNG BRYNGELSON, Minneapolis: Before discussing the major aspects of the treatment of the 127 cases reported at the close of the paper, I should like to point out that the success of the dominance technic is so marked that I have come to feel that a differential diagnosis, in which other causation factors in regard to treatment are usually considered, though valuable, is not essential. Oral erotic symptoms, lack of visual imagery, consonant or vowel interference, psychoneurotic trends or a dozen other factors around which different treatments have been built need not be considered as such in cases in which stuttering is definitely traceable to a shift from left-sided to right-sided cerebral dominance. I am suggesting the possibility that no matter how a stutterer stutters, nor for what etiologic reason, he can be treated effectively by the method based on the theory of cerebral dominance.

MISS MILDRED A. MCGINNIS, St. Louis: It was with a feeling of pleasure that I read the abstracts of the papers contributed to this program, and found that the true psychoanalysts were absent and even such tendencies were in the minority. I have not the opposite opinion that behavior analysis has no place in the therapy for the relief of stuttering, but I do believe that the erudite premises and conclusions of a number of such theorists have been a greater pleasure to their student minds than a help in affecting a desired result.

DR. G. HUDSON MAKUEN (quoted): That many stammerers are profoundly neurotic must be admitted, but the question is as to what extent their neurotic condition is the cause of the stammering and its continuance, and to what extent it is the result of the affection. That it is more largely resultant than causative, at least in the chronic cases, is a conclusion that has been reached after a careful study of this subject, and the only way to rid the stammerer of his nervousness is to prove to him that he can overcome his stammering. The treatment of the affection, therefore, must be approached through the physical, rather than through the psychical condition of the patient, because the one dominating and overpowering idea is that of his inability to conquer his defect.

DR. C. S. BLUEMEL, Denver: Emotion has long been suspected in connection with stammering. It was first thought of in simple terms, such as embarrassment

and self-consciousness. Later the study became detailed, and the stammerer's sublimations were said to have gone awry. Let us meet this contention by assuming that our psychoanalytic friends are right, and that the problem is one of conflicts and psychic traumas. Then the question arises, Why stammering? Why does not the patient develop facial tic, torticollis, cardiospasm or astasia-abasia? If we admit that emotion accounts for the presence of the nervous disturbance, then we must look for something that determines the character of the disturbance. In the case of stammering, we must go beyond the emotion and investigate the verbal thought. These facts seem to point away from emotion and in the direction of mental speech.

Conditional:

Dr. Paul L. Schroeder, Chicago: While these statistical calculations indicate the type of psychoneurotic, unaggressive behavior generally associated with stammering, they do not of themselves tell which is primary and which is secondary. It may be that stammering is a symptom of this type of personality defect, or it may be that this type of personality is the result of the child's reaction toward his stammering. Or it is possible that the stammering and the personality of the stammerer are both symptoms or concomitants of some deeper-lying causal complexes which will require more intensive research to isolate and identify.

Dr. Elmer L. Kenyon, Chicago: I grant freely that the muscular disturbance is dependent to an important degree on emotional excitement at the moment of the production of speech. When the disordered, muscular phenomena are not dependent on emotional excitement, they are then dependent on either psychomuscular habit or on intentional production by the stammerer of muscular wrongness of action. But I do grant that the nervous phenomena as manifested cannot be dissociated from the muscular phenomena any more than the muscular phenomena can be dissociated from the nervous phenomena. In other words, as the muscular disturbance is dependent on the emotional excitement, so also are the emotional manifestations just as definitely dependent on the peculiarly disturbing action of the speech mechanism on the mind of the stammerer.

The Rôle of Sinistrality and Ambidexterity as a Causative Factor in Stuttering

For:

Dr. Travis was the most earnest advocate of the "left-handed" causation. He was supported by his disciple Mr. Bryngelson, by Miss Liljegren and Dr. Stivers, and by incidental references to the subject by Dr. Blanton. Miss Liljegren, one of the disciples of Dr. Swift, famous for his theory of visual imagery, includes in her list of probable causes of stuttering among her own pupil-patients a "change from left-handedness to right-handedness."

Dr. Charles G. Stivers: The brain center governing speech, which is expressed by the lips, tongue, cheeks, larynx, etc., merges with the centers governing the leg and arm and hand. Changing neural dominance of handedness from the left to the right side of the brain therefore affects the centers for speech and for the leg, and confusion, often followed by stammering, results from interference with the native pattern.

Dr. Lee Edward Travis: As we have discovered from the study of cases, a stutterer's present motor leads may be out of harmony with determined native physiologic preferences. Since in many cases the stuttering dates from the beginning of such a discrepancy and disappears with the removal of it, we are led to consider in every case the possibility of changing or reverting in motor leads to the seemingly nonpreferred hand. When it is decided that a "right-handed" stutterer should become left-handed, it is necessary that he make the change as complete as possible. Every manual activity in which a lead from one side of the body is possible should be controlled from the left side. Such would include writing, throwing, cutting, eating, dressing, handling a tennis racket, etc.

Against:

MISS MILDRED A. MCGINNIS: I believe that only when a shift of handedness has been tried out to a 90 per cent satisfactory result will theories of its effects on stammerers be justified. By a satisfactory result, I mean a correction of at least several years' duration. In my experience, I have found few stammerers to be, or to have been, left-handed, while there are many people who have been retrained to use the right hand with no ill effects. This year we have had two left-handed patients with oral inactivity whose articulation and behavior were in such a state that no harm could be done by experiment. While both of these persons are now using the right hands with facility, improvement in their speech is no more or less rapid than others who have been trained without attention to handedness.

MR. FREDERICK W. BROWN: Wendell Johnson's "Because I Stutter," with its introduction by Dr. Travis, forms an interesting and valuable document. I cannot agree, however, with Johnson's statement, apparently supported by Dr. Travis, that "a thorough psychoanalytic investigation of his life . . . showed quite conclusively that there were no psychopathic or mental or nervous causes underlying the defect," nor with his conclusion that "as soon as I have succeeded in shifting completely to left-handedness, I shall talk without stuttering." Why may not the psychoanalytic treatment as well as, or instead of, the training in left-handedness have been responsible for his improvement to date, and why may not the gradual reintegration of his personality and his readjustment to society, resulting from his psychoanalysis, be responsible for any future improvement in his speech? Again, it has not been shown that the training in handedness or other methods employed to establish the dominance of one side of the cerebral cortex are the sole or even the predominant causes of the improvement already made. In fact, Johnson's narrative furnishes abundant evidence that such factors as the continually reiterated positive suggestions of his instructor, the instructor-pupil relationship and the physician-patient relationship, and the restoration of, or increase in, self-confidence and insight, may have been responsible for much, if not all, of the improvement shown.

THE VALUE OF SPEECH DRILLS

For:

DR. ELMER L. KENYON: The bases of my own conception of the treatment of stammerers go back to G. Hudson Makuen and Herman Gutzmann, but especially to Makuen. Dr. Makuen taught conscious active control of the chest, coupled with consciously directed production of each elemental sound of speech.

DR. J. HUDSON MAKUEN (quoted): Stammering in the great majority of instances is due primarily to faulty or delayed phonation, or to a lack of promptitude in the vocal mechanism. Voice culture, therefore, should be the keynote of all methods of treatment. The first essential of speech production is adequate voice production. The restoration of good speech consists largely in the development of a more vivid and distinct auditory imagery for speech sounds.

MISS MILDRED A. MCGINNIS: Speech drills must not be drills in name only. They should be component parts of words, such as syllables composed of consonants and vowels for the purpose of establishing a good voice, the habit of making the correct coordinations for all consonants and the habit of taking time to give each syllable its value. When syllable drills can be given with such requirements, there should be an immediate correlation with speech, and conversation relative to mental hygiene should be carried on in a new and better voice. In detail, speech drills would include attention to vowel quality in words, articulation of final consonants and attention to pauses, as the case requires.

DR. CHARLES G. STIVERS: Whispering, soft tone, inflection, normal tone and loud voice are all used in their places. I put great stress on inflection and show by the effect of my voice on my dog that it is the inflection that attracts or repels.

Conscious articulation, with the making of large molds or positions for the various sounds, is of importance. I use here a form of massage to loosen the muscles of the lips and cheeks, which are often very tense. I have never put my fingers into the mouth, but sometimes I place a wooden applicator on the spot where the sound is made.

Miss Clara B. Stoddard, Detroit: The Reeds, whose methods I use, taught phonetics to lead the pupil out of his old ways of attacking speech, and to introduce him to new methods that should eliminate hypertonicity, juxtaposition of the organs of speech and the attempt to produce two sounds at the same time. They built up stronger verbal and visual images of consonant and vowel sounds, training through tactual, auditory, visual and association areas.

Dr. Elizabeth A. McDowell, New York: The third step is a more or less hit-or-miss scheme for experimenting on his own part with exercises and schemes for gaining success in acquiring the desired speech reaction. These must be very simple at first. We arranged them into a hierarchy of stimuli or speech exercises according to their frequencies of success. One exercise that is universally successful is speaking isolated syllables in unison with the instructor who is employing an easy, well-modulated voice.

Dr. Lee Edward Travis: The third period consists of speaking and copying at the same time. The individual begins to write each word before he speaks it, thus having the writing movements precede each spoken word.

Mr. Bryng Bryngelson: Make words your hobby. When you are writing and talking remember to talk loud and to keep your speech muscles in good working order by constant exercises.

Write pages and pages of your "Jonah" words. Write them out in full, speaking them as you write them.

Practice voluntary clonic spasms in the reading of simple material. All stutterers, whether their natural spasms are clonic in type or not, begin with this stage.

Against:

Dr. Lee Edward Travis: Although phonetic drills are certainly not indicated in the treatment for stuttering, certain exercises in ordinary and interpretative reading, reciting poetry and singing may be helpful.

Dr. Smiley Blanton: As to phonetic training, it is unwise and makes the stutterer most self-conscious of his speech. Practice in breathing may give the stutterer some relaxation, but if such exercises lead him to think that his stuttering is caused by defective breathing and take his attention from the psychologic factors, they will do more harm than good. Vocal exercises, such as are used to improve the voice of nonstutterers, and exercises, such as are used in any good class of voice training, may be used, but the stutterer should not be told that this is to cure him of stuttering.

Dr. John M. Fletcher, New Orleans: As a student of this disorder, I have lodged criticisms against the diagnosis of stuttering as a mere physiologic incoordination induced by wrong habits of speech, and hence against the phonetic drill methods of treatment that are based on such a diagnosis.

The Use of Suggestion in Attempting to Induce Better Speech in the Stutterer

For:

Mr. Samuel D. Robbins: The subconscious fear of stammering may be overcome by asking the pupil every night before falling asleep to picture the most confident person he knows speaking fluently to his old friends, and then to picture himself doing likewise.

Dr. Smiley Blanton: The application of the best principles of child guidance should be put into practice in the home. Under mental hygiene is included the

methods of psychotherapy, suggestion, persuasion and mental analysis, psychoanalysis being a special type of mental analysis.

DR. CHARLES G. STIVERS: Replace the "I can't" by the "I can." In this, show the patient how pressing his palms together will make him conscious of the effort. Tell him to remove one hand, which is the "I can't" idea, and the "I can" idea remains. Explain that it is necessary to drop the sense that there is any blocking or damming of the ability to speak.

Take any word over which the patient stammers. Find out why he feels that he has to stumble. Show him that the wrong feeling toward it is the reason why the word bothers him, not because any more effort is required to pronounce this word than any other.

Against:

DR. ELMER L. KENYON: Early in the period of training, the effort at conscious control is applied to the act of talking. Gradually this consciously controlled effort is developed until speech production has become normally smooth, excellent in quality and attended with little or no hesitation. In order not to be misunderstood, I will state that "suggestion" has as little part in this training as it does in the teaching of proper management of the body in learning to swim.

General Comment

DR. G. OSCAR RUSSELL, Columbus, O.: I hesitate considerably to discuss theories as to cause. Everybody will have observed that most of the various schools of treatment may be credited with certain effective results. A careful scrutiny of the therapy used by these groups espousing many and varied and often conflicting theories will show that the methods of treatment have much in common in most cases. Furthermore, in no particular case that I know of is that therapy based entirely on the theory in question. All of us owe each other and those who have gone before us a great deal.

DR. ELIZABETH A. MCDOWELL: Perhaps the most outstanding obstacle to real progress in improving our technic for correcting stammering is our tendency to oversimplification of the problems involved. We are seeking the alchemist's stone which will reduce all of our hunches to golden facts with one experimental effort. Our literature is devoted largely to espousals of theories explaining this or that alleged cause of the phenomena and prophesies that the removal of this cause will bring permanent cures. For this reason, our field has long been fertile ground for the "quack" or the cure-all specialists. Many of our most skilful therapeutists will practice their arts of speech improvement with admirable results and discriminative effort, and then they will sit down in arm-chairs and weave explanations of the reasons for their success into a logical romance with some one attractive notion as the hero.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Meeting, Jan. 5, 1931

GEORGE DENNIS, M.D., *President, in the Chair*

NORMAL INTERIOR LARYNGEAL FUNCTIONS WHICH BLOCK THE VIEW OF THE GLOTTAL LIPS (MOTION PICTURE DEMONSTRATION).

DR. G. OSCAR RUSSELL, director of Phonetics Laboratory, Ohio State University, Columbus, Ohio.

The pitch used by the subject during articulation radically affects the view of the vocal lips. Very low pitches cause so-called "retracted epiglottis." The pulvinar is pulled toward the cartilages of Wrisberg, causing postero-anterior closure. Excessively high pitched tones or "tight" or stringent voices cause

constriction of the lateral interior part of the larynx hiding all but a mere edge of the vocal lips. Some vowels expose a view of the full length of the vocal lips; others shut off the view progressively more and more. The interior of the larynx closes (anteroposteriorly) more and more, thus impeding the view in a progressively increasing way as one passes through the vowel series: i-peep; I-pip; ɛ-pape; e-pep; ae-pap; ə-pup; a-pop; ɔ-paw; o-pope; u-poop.

The x-ray pictures published in my "Speech and Voice" show that the front cavity for "ee," (i) is hardly as big as one's finger, and it is therefore impossible to insert an ordinary laryngoscope through it for any kind of a view (as one can my "Nongag glottoskop" or my "Fonofaryngoskop") without enlarging that anterior buccal cavity more than the vowel will permit. If the cavity is enlarged, the vowel changes to the following: First, usually to the vowel "uh" (ə) as in "pup." It is true that part of the cords are then visible, but they look dumpy. The anterior half remains hidden. Second, to the vowel "ah" (a) as in "pop" producing a "retracted epiglottis." No cords are visible. Third, to something between (I) as in "pip" and (ɛ) as in "pep" when more of the cords will be visible. In other words, roentgenograms of the vowel "ee" (i) "peep" show the largest distention of the pharynx and the smallest distention of the mouth. Hence, if proper apparatus is available that is the best vowel to use in order to avoid impediment of the view of the vocal cords.

A guttural voice closes the false cords and hides the vocal lips. Gagging causes both posterioranterior and lateral closure. A gagging or interrupted voicing closes the false cords above the vocal lips. A cough closes the ventricular bands above the vocal lips, thus hiding them. Clearing the throat closes the false cords as well as the vocal lips.

Hence, it is absolutely necessary that the subject articulate on his normal easy range speaking tones, with relaxed free quality, if an unimpeded view is to be had of the vocal cords throughout their whole length and of the entire interior of the larynx.

DISCUSSION

DR. LEO KALLEN: That intralaryngeal activity, per se, other factors supposedly being equal, exercises an influence on, or rather that it actually is one of the conditions on which quality and character of voice depend, is a fact that has been known for several decades, beginning with the classic attempt of Czermak, in 1860, to photograph laryngoscopic images, which were successful enough to depict the cords in phonation, the shape of the glottic chink and the relation of the ventricular bands.

With the development of photographic technic, depiction of the interior of the larynx in its varying phases of function made corresponding progress. However, it is really only in the last two decades that technical skill has made it possible to depict the larynx with an ever decreasing quantum of imperfection.

Dr. Russell has given an excellent demonstration of a motion picture of intralaryngeal activity synchronized with its vocal effect. This is an important achievement, not only because of a faithful portrayal of a process otherwise hidden, except to the eye of a phonendoscopic observer, but because synchronization with sound offers a new method of study and instruction. Those of us who are concerned with the diagnosis and treatment of functional disturbances of voice are in need of more and more information concerning intralaryngeal processes in connection with vocalization. Dr. Russell's success in synchronizing these two in the form of a tonal motion picture is an important step in that direction.

DR. ELMER L. KENYON: Somewhat more than a century ago the present national movement for the education of the deaf in speech and lip reading was inaugurated. About twenty-five years ago a national movement for the educational treatment of disorders of speech and of hearing was likewise inaugurated. Last week the fourth national convention of the American Society for the Study of

Disorders of Speech, an organization that is attempting to guide this newly developing movement, was held in Chicago. This newer movement should have the sympathetic cooperation of the medical profession.

By its very nature, the ordinary laryngoscope cannot show the interlaryngeal movements without interference with free action of the vocal cord excepting in certain patients for the sounds "ah" (are) or "aw" (awed), and, in addition, it cannot show most of the sounds of the language at all. Killian was instrumental in bringing the larynx into direct view, but with practically complete suppression of movement. Dr. Russell, following with his mechanically acute mind the more basal work of his predecessors, has opened up for us in two ways an almost unhampered view of the interior of the larynx in action for any sound. These new electrically lighted laryngoscopes are of real value to the laryngologist, and will in time become a part of his armamentarium and of that of our medical schools. They can be used equally well by nonlaryngologists who are interested in disorders of speech. Dr. Russell receives no personal profit from the sale of these instruments.

Now we are enabled to witness further fundamental progress in the disclosure of the interior of the larynx in action. These interior movements are disclosed in detail and with clearness as motion pictures. About twenty years ago, working from the standpoint of the extrinsic movements of the larynx, I was able to point out that each basal sound of the language must require detailed differences in the approximation of the vocal cords. It is now of great interest to me to have the accuracy of my observations proved by these motion pictures of the interior of the larynx in action.

The interpretation of the significance of the movements, as disclosed, will require much time and study. Exactly what movements serve to produce a certain type of sound, as to language significance, musical quality and pitch, must be gradually determined. This information will be a companion to the evidence of the sound itself, which we have always had. But these facts, even together, cannot solve the problem of developing the artistic voice. That problem concerns the question of so controlling the musculature of speech production as to attain any desired vocal result. Just how these pictures are to forward the solution of that problem is yet to be determined.

The complexity of the movements within the larynx is remarkable. Did you ever think that the larynx gives the brain no knowledge of what is going on within it? There is no way in which the brain can directly understand what is going on in the larynx except in the vocal result. In order to realize the complexity of the act of voice production, we must think of the movements of the extrinsic muscles of the larynx, which also are complicated, as well as of the intrinsic muscles. The extrinsic and intrinsic muscles, are controlled in action for voice production by an automatic cerebral mechanism, which acts indirectly in accordance with the conscious purpose of the individual to produce a definite sound. The problem of voice development consists in the determination of exactly how to gain control of these muscles in order to produce any particular vocal result at will. The motion pictures throw new light on how the larynx produces particular vocal sounds, but they do not solve the problem of how to control the vocal mechanism in order to attain to particular sound results. What they may ultimately contribute to this practical purpose is a matter for the future to decide. That they may be developed into a diagnostic procedure of much importance in certain doubtful cases seems highly probable.

Dr. Russell tells me that he has already made 1,000 motion pictures of the interior of the normal larynx, largely in singers; and thus is being opened up a large new field of study.

DR. ROBERT SONNENSCHEIN: Dr. Russell has done marvelous work. It seems to me that we have in these pictures a proof of the fallacy of the former assumption that in the movements of the larynx during swallowing the epiglottis folded backward and closed the glottis. These pictures do not show the act of swallowing, but I take it that in some of the movements of speech the motion is

almost the same as in swallowing. For a time it was thought that the movements of the arytenoids helped to close the larynx during swallowing. Then again, a few years ago, Dr. Mosher presented an excellent paper in which he stated that from x-ray studies he assumed that the epiglottis covered the larynx in the act of swallowing. I think that in these pictures we see that it is the movement of the arytenoids and the false cords that serve this purpose.

DR. ALFRED LEWY: I do not see how these pictures can show one thing very well, namely, the up and down movements of the larynx which occur during phonation. I do not know how Dr. Russell can show that in addition to the anteroposterior and lateral movements, but perhaps he can devise something that will also show those movements.

Also, in the use of the laryngeal mirror we do not find that in most cases the attempt to phonate the vowel "e" gives us the best view of the interior of the larynx, but, of course, conditions are changed by our traction on the tongue. Sometimes the "hi" and sometimes the "e" is better. That is another demonstration of the fact that by using traction on the tongue as we have to we are disturbing the normal relation of the larynx.

I wish to take issue with Dr. Kenyon concerning one matter: I have always felt that the patient was always conscious to a certain extent of the movements of the larynx by something like kinetic sense, that we are not dependent entirely on audition. That has been told me repeatedly by singers, who have said their vocalization "did not feel right." This has impressed on me that there is some sensation akin to kinetic sense in addition to the auditory sense.

DR. ELMER L. KENYON: I agree with Dr. Lewy, however, there is no question but that the statement I made is essentially true. We get these movements in the larynx primarily by thinking of the sound. However, there is a distinct sensation related especially to the mold of the mouth required for the sound, and, as I believe, to the action chiefly, not of the vocal cords themselves or of their muscles, but to the extrinsic muscular movements. Undoubtedly such sensations are of great value to singers, and they also serve primarily to enable the deaf person to acquire speech.

DR. J. HOLINGER: Years ago I was in a position to amputate the epiglottis on account of tuberculosis, often down to the lowest ridge, and no loss of function could be noticed. The swallowing was often greatly relieved, and no food entered the larynx. I came to the conclusion that we overestimated the importance of the epiglottis. Patients with such conditions now go to tuberculosis institutions, as soon as we make the diagnosis, and we do not hear any more of them. A large amount of material must accumulate there. So far, in our Society, I have not heard of any scientific contribution on the subject that would be adequate.

DR. G. OSCAR RUSSELL: The pictures shown here form a part of a larger study financed by the Carnegie Foundation and conducted in collaboration with members of the American Academy of Teachers of Singing and of prominent opera and other vocal stars of this country and abroad. The purpose was to establish the physiologic causes of differences in the quality of the voice. The major part of these experiments consisted of x-ray pictures taken at a high rate of speed. A large number of subjects were utilized including Althouse, Bori, Gigli, Johnson, Tibbett and many others of recognized vocal ability. By our x-ray technic we have been able to devise a set-up that makes it possible to compute the exact length of the vocal cords; likewise, to indicate the rise and fall of the larynx, the opening and closing of the velum, and dimensions of the vocal cavities themselves and of the openings, including those of the lips. All of these may be measured accurately to a fractional part of a millimeter. It will be seen, therefore, that the best information bearing on the question asked would be obtainable from our x-ray studies rather than from the moving picture studies of the vocal cords presented tonight.

The only way we would have of ascertaining whether the larynx rises or falls by study of these motion pictures of the vocal cord (since they are from

a superior laryngeal position) would be by observing the field and also by studying the changes in focus. That would not be anywhere near as reliable as the x-ray pictures in question.

As to the point raised by one other question, I do not believe it is possible to find a subject who will be able to articulate a clear vowel "ee" during the old style laryngoscopic examination in which the ordinary laryngeal mirror is inserted and the light is reflected in by means of a condensing head mirror, or by some of the more recent laryngeal mirrors which attach a light at the distal end of the handle immediately in front of the laryngoscopic mirror. The reason is obvious when you recall the x-ray pictures included as a part of the demonstration tonight. For the vowel "ee" requires a very small buccal opening, normally no larger than your finger. And since this curves upward, it is not possible to see through it into the posterior part of the pharynx. When this question first arose, I verified that observation by attending a large number of examinations by different doctors where the attempt was made to get the subject to articulate a clear "ee." I believe I could say that though the subject often thought it was the vowel he was producing, something approximating "uh" was invariably produced in lieu of the "ee." I stress this fact to get your reaction as you go back and listen carefully for the sounds you hear, and I should like to know that reaction, particularly as you may obtain it from your office assistants, nurses and strangers who are not expecting any particular sound. I dwell at length on this question, because this demonstration of films makes it clear that a vowel "ee" pronounced with unmistakable quality is necessary if a complete view of the interior of the larynx is to be had.

I was much interested in Dr. Kenyon's note to the effect that he had assumed long ago from exterior palpation and other methods that the larynx changed position rapidly in passing from one vowel sound to another, because when I published in my book, "The Vowel," the first still pictures of the vocal cord in unhindered vowel positions, I was taken severely to task by a few who insisted that the vocal cords did not look so long as they appeared in my pictures. The reason is obviously because that in all pictures taken formerly the ordinary laryngoscopic mirrors were used, resulting, as I have just pointed out, in necessity for the subject to articulate a vowel "uh." As you noted, this vowel "uh" invariably blocks off a substantial part of the view of the vocal cord. It is not possible to see the anterior commissure and the area well up under the pulvinar. You will have observed that these motion pictures verify the earlier still photographs. In all cases in which a clear vowel "ee" is articulated, the full length cord is shown in its stretched out appearance. And the whole interior of the larynx as well as the pharynx is seen to change as one passes from vowel to vowel.

I agree with Dr. Kenyon that my "Nongag Glottoskop" is the most valuable of all the laryngeal pieces of apparatus I have devised. You will notice that the subject may close his lips and can articulate practically any speech sound. Its usage with an "m," therefore, makes it possible also to obtain a sharp, clear view of the whole nasopharynx viewed from beneath, so that in examinations for adenoids it likewise has distinct advantages.

THE PRACTICAL VALUE OF THE AUDIOMETER. DR. G. HENRY MUNDT.

The audiometer is a practical mechanism for making graphic records of hearing by which the future progress of the patient may be judged. It is the only mechanism by which a reasonably accurate percentage of loss of hearing may be determined. The audiogram is the only practical graph by which a patient may be watched by different otologists. A satisfactory audiogram can be made in many patients who seem unable to cooperate sufficiently for good fork tests. This is especially true in children. There are other methods superior to the audiometer for testing the lower tones, but the audiometer is the best mechanism for testing the upper portion of the scale. Defects in the upper portion of the tone scale call for a careful general examination of the patient. An audiometric study should be made in every patient who complains of defective hearing.

DISCUSSION

DR. LOIS D. GREEN: In collaboration with Dr. Shambaugh, I am attempting to clarify some of the factors of bone and air conduction as recorded by the 1-*A* audiometer and thus give added practical value to the examination.

The customary audiometer curve I shall designate as the standard curve, and it is taken with the head phones in contact with the head and cushioned with sponge rubber. This is the curve ordinarily designated as air conduction. In cases of otosclerosis or other conduction deafness, the picture given by this curve differs markedly from that given by the whispered voice and tuning forks by air conduction. In order to give a truer picture of the loss of hearing by air conduction in these cases, I began taking audiograms, holding the receiver as near to the auricle as possible but without any contact with the head. These I designated pure air conduction curves, and they were made on normal subjects, patients with conduction deafness and those with nerve deafness. They were plotted on the same charts with standard curve (phones in contact) and bone conduction curves made by the Western Electric bone conduction receiver No. D 80904.

In the course of a year certain points have become apparent. The curve for pure air conduction in normal subjects is from 20 to 30 sensation units lower than the standard curve for frequencies up to 1,024 double vibrations or more usually 2,048 double vibrations, when it becomes only about 5 units lower and parallels closely the rest of the standard curve.

In a true conductive lesion, the pure air conduction curve is from 40 to 60 units lower than the standard curve for the lower frequencies and again approaches to within from 5 to 10 units of it at 2,048 double vibrations. This gives a truer picture of the useful hearing of a patient with a conductive lesion. In a true nerve lesion the pure air conduction curve bears the same relation to the standard curve that it does in the normal ear.

When the bone conduction receiver is used the results are apparently not reliable beyond 512 to 1,024 double vibrations, unless the patient is very deaf. At these points the instrument is emitting so much sound by air that the curve is almost the same whether the apparatus is in contact with the head or slightly removed.

In cases of distinct conduction deafness, the curve produced by the bone conduction receiver may rise above the standard and pure air conduction curves and approach that of normal hearing. In cases of nerve deafness, it bears the same relation to the other curves that it does in the normal, being from 20 to 40 units below the standard curve at from 32 to 64 double vibrations and about 60 units below at from 512 to 1,024 double vibrations.

These observations suggest that the standard curve with the phones in contact with the head does not represent the true air conduction, but that a true air conduction curve will have to be developed and standardized. They are also of aid in visualizing and recording more accurately various type of deafness. Possibly additional light will be thrown on the Rinné and Schwabach tests, particularly if the work can be done in a sound proof room.

DR. G. OSCAR RUSSELL: I think that Dr. Green's contribution is vital, and I believe that much more work of this sort is needed. In one of the papers I presented before this Society last year, I showed a hearing test graph which could advantageously be used in lieu of an audiogram. It is used as is the latter with any Western Electric 2 *A* or 1 *A* audiometer, but has an advantage over the audiogram in that it shows at a glance the exact status of the subject's hearing in terms of the amount of hearing remaining and lost for each tone and the whole or any part of the range, so that the median or peak is also graphically portrayed without any computation or reference to a conversion table. These advantages over the audiogram need no comment. I believe that Dr. Bunch and Dr. Shambaugh, who had protested at some rather loose usage of the computation of loss of hearing on the audiometer, are justified. The former has made a particular point of the fact that the customary statement of loss of hearing is not adequate. One principal reason why this is true is that the term represents a too vague

generalization. This is particularly so when one follows the recommended procedure of choosing three tones in the middle of the spectrum and averaging these with an arbitrary correction for a statement of what hearing is remaining in the subject. We long since observed that pedagogically, at least, all kinds of false conclusions are thereupon drawn. It is admissible that this statement is infinitely superior to what was formerly available, but surely we should not be content with anything less than the best. Since it is inadequate, it would seem advisable to cease the usage of this statement of loss of hearing. There may be times when a statement of the median loss through the whole audiometer range would suffice, and might stand as a statement of the individual's hearing status, but the question arises in that connection whether it would often not be better to use the peak loss as a criterion by which to judge. Certainly it is true that for pedagogical purposes it is often absolutely necessary for us to know whether the loss is in the low frequencies or high frequencies, and the teacher must of necessity know whether the loss in the low frequencies is serious. Over 40 per cent of the meaning of speech is carried in a rise and fall in intonation which involves the area from 60 to 256 cycles in male voices, and this is precisely the area that is left out of consideration in the usual statement of loss of hearing computed on the basis of the three tones previously mentioned. On the other hand, if the teacher is engaged in correcting difficulties of speech, it is quite essential that she know the status of the person's hearing over the range from 3,000 to 8,000 cycles, for this is precisely the range involved in the characteristic tones required for the production of such sounds as "s," other fricatives and even most unvoiced stops.

The physician cannot be blamed for not desiring to waste the time necessary in order to refer back to a conversation table and compute the loss of hearing for each individual tone. This is the principal reason why we prefer the aforementioned hearing test graph in lieu of the audiogram. It shows everything the audiogram does, and at the same time indicates at a glance just exactly what the loss of hearing is for any particular range. It can be used for either the 1 *A* or 2 *A* audiometer. The patient who takes such a graph with him is better directed, and this is particularly true of children whose teachers need to know what can and cannot be done in the matter of training and retraining the sound patterns involved in a cerebral loss of hearing, which must inevitably follow a functional loss if nothing is done. I believe otologists generally could render a great service to the people of this country if they would all give their full support to recent moves they have fostered which tend in that direction. I refer particularly to those sponsored by Dr. Hayden and others behind the work of the League for the Hard of Hearing.

I wish to call attention to the phonograph speech tests now available through the work of Dr. McFarland, since through their use considerable information may be added to our present stock of knowledge in regard to the cerebral deafness.

DR. G. W. BOOT: I remember some years ago hearing a highly scientific paper read before the Chicago Ophthalmological Society on the proper method of illuminating a test card. The essayist proved by calculus and demonstrated by means of a graph that the only correct method of illumination was by means of a parabolic mirror.

In this highly scientific age, if an article is not illustrated by one or more graphs it is discredited, and if the author of a paper furnishes the requisite number of graphs it is accepted as scientific. I often wonder how Darwin managed to put his great work across without a graph to illustrate it.

The audiometer has two of the modern requisites; one always has a graph to show and one uses that modern method devised by Henry Ford and introduced into medicine by the famous clinic that habitually introduces its papers by the statement "in our last 10,000 cases," or words of similar import—mass production.

DR. ROBERT SONNENSCHEIN: I always hesitate to say anything about the audiometer because the impression has gone abroad that I am prejudiced against it, but this is absolutely not so. I think the audiometer has certain advantages, and feel that Dr. Mundt is to be congratulated for his fair way in presenting it.

Most persons say we cannot practice otology without it, forgetting that innumerable persons were examined successfully before it was devised. In many ways it is practical from the standpoint of speed, ease of manipulation, etc., as compared with other methods in vogue.

First, let me call your attention to the fact that while the audiometer is an instrument of precision, and I have always been in favor of such devices and methods, the various types of audiometer are not calibrated alike. The Western Electric is best known and most widely used. It is calibrated so that it tells the loss of hearing in sensation units, and to get the percentage of loss of hearing you multiply the number obtained by 0.8. But the calibrations of the Jones-Knudsen, of the Krantz, the Dean-Bunch and the Gutman audiometers (the latter it seems was the first one to use the vacuum oscillator tube in connection with the audiometer) are entirely different. I do not know about the physics of the subject, but I am told that the curves or graphs of the different apparatuses are not comparable. It is true that it is nice to have graphs to show the differences in hearing at various times, but it is well not to forget that no less an authority than Harvey Fletcher stated that if we have the so-called "constant" of damping we get the same data regarding loss of sensation units with tuning forks that we do with the audiometer. The calculation is simple and he gives the formula. So if you have four, five or six calibrated forks you can get the same curve as with an audiometer. While I have always advocated that we should excite forks uniformly, it has been shown that the decrement or rate of damping of the fork is always the same even if there is some difference in the force used. The formula is loss of sensation units $= \Delta\ (t\text{-}t')$. Δ is the constant of damping; $t =$ normal hearing time; $t' =$ hearing time of patient. Let us say the decrement for the small a-1 is 1.5; the time the patient should hear it is 100 seconds; if you find the hearing is only 70 seconds you simply substract this from the 100 and the difference is 30, multiplying this by 1.5 gives 45 sensation units loss. If you have the forks you can get the same graph that you do with the audiometer, but if you use many forks it takes a little longer. You can get the same graph you get with the Western Electric for it is calibrated by the same system, so there is no reason for saying that we cannot get the same graph with the forks as with the audiometer.

I must take exception to Dr. Mundt's statement that the Bezold forks are the only ones with which tests can properly be made, for I am sure you are aware of the work of our committee of the American Academy of Ophthalmology and Otolaryngology, and know that the forks prepared by Mr. Eisenbaur of the Riverbank Laboratory at a much lower price give the same data. I do not say this to deprecate the Bezold forks, but one can get the same results with the new magnesium alloy forks, which are rust and tarnish proof and weigh only one-third as much as steel.

Dr. Mundt quoted from Dr. Shambaugh's excellent paper which was read in Copenhagen. He stated that for the lower tones the audiometer is of no particular value; for the middle tone it is perhaps superior to the forks, and for the highest tones he thinks it is better than the forks. Recently Mr. Eisenbaur, of the Riverbank Laboratory, devised sounding rods of a pitch of c^5 (4,096 double vibrations) which vibrate from fifty to seventy-five seconds, whereas the best tuning fork of that pitch rarely vibrates longer than thirty seconds. He also perfected a rod of the pitch of c^6 (8,192 double vibrations). It is quite true that the earliest evidence of a change in the nerve of hearing is in the appreciation of the high tones, but the exact amount of hearing does not make any essential difference; all you need is an approximate idea. If the loss of hearing is marked, one can tell it in the course of ordinary conversation. By using the residual air in testing with unaccentuated whisper or conversation, one gets a practical estimate of the patient's hearing. Dr. Shambaugh also has pointed out the fact that you do not need any apparatus for this purpose. The audiometer is a good apparatus; it is fine for the study of the physiology of hearing and for research, as Dr. Shambaugh has shown, but I still contend that with a few good

forks, and tests properly made, one can get a good practical idea of the degree of hearing ability. Dr. Green demonstrated her work to me this afternoon, and while telephone men tell us that we get only air conduction with the receiver against the ear that is not entirely true, for we get some bone as well as air conduction. We know that if we hold the receiver against the ear we hear better than when it is out of contact with the auricle.

DR. G. HENRY MUNDT: I think this type of discussion should continue; it is very wholesome. I do not stand before this organization as an advocate of the use of the audiometer.

Taking up Dr. Green's discussion, the standard graph undoubtedly is valuable. That will be worked out, and I have no doubt many of us will have audiometers which are far superior to this *2 A* audiometer. Such work as Dr. Green is doing in an effort to get pure air conduction is valuable. From a purely practical standpoint with one patient, I believe that the graph made today and made again in a few weeks is valuable. Sound proof rooms are not easy to produce in Chicago. I have tried it, and think Dr. Green will have difficulty, unless she gets some distance from the ordinary units of transportation. There is no question that hearing tests of any kind should be made in as nearly a sound proof room as possible. I am perfectly willing to admit that when one figures the loss of hearing on an audiogram one has not a definite method. Loss of hearing is, I think, better measured in the average individual by the voice test, but in the evaluation of progress in a patient the audiometer is superior.

I appreciate Dr. Sonnenschein's discussion, and if he reads my paper he will find that I said that as a minimum type of fork we should have the Bezold. I was trying to eliminate the use of two or three small forks which are of little value: to be more specific, the set commonly sold as Hartmann's tuning forks. I said in this paper, and I have always maintained, that in determining the loss of hearing the voice test is probably more valuable than the audiometer, and yet for comparison from time to time the audiometer is the more valuable.

Book Reviews

ATLAS DE ANATOMIA DEL ORGANO DEL OIDO. By DR. PEDRO BELOU. Buenos Aires: Estudio Grafico "Oceana," 1930.

Dr. Belou has presented in the form of an atlas a comprehensive and detailed study of the anatomy of the organ of hearing, in which every phase of its intricate anatomy has been included in anatomic preparations reproduced by illustrations. The material consists of 236 loose-leaf cards containing illustrations, many of which are prepared to be studied with the stereoscope. The volume represents a tremendous amount of work and will constitute a valuable addition to the library of those who are interested in something more than just the surgical side of otology. The work does not include in any detail a study of the histology of the internal ear.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gabriel F. Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: New Orleans. Time: 1932.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. F. Barnhill, Miami Beach, Fla.
President-Elect: S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: French Lick, Ind. Time: Sept. 14-19, 1931.

American Bronchoscopic Society

President: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Place: Atlantic City, N. J. Time: June 15-17, 1931.

American Laryngological, Rhinological and Otological Society

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

American Otological Society

President: Dr. David H. Walker, 101 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Harry P. Cahill, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. Lyman G. Richards, 319 Longwood Ave., Boston.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Albert E. Hillis, 1115 Pacific Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

---

* Secretaries of societies are requested to furnished the information necessary to make this list complete and to keep it up to date.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. Claude T. Uren, City National Bank Bldg., Omaha.
Secretary-Treasurer: Dr. Frederick H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Fontanelle Hotel, Omaha. Time: November, 1931.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fletcher D. Woodward, Box 162, University, Va.
Secretary: Dr. W. D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: New Orleans. Time: November, 1931.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

STATE

COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. E. Cooper, 227, 16th St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. William L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasurer: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Gordon F. Harkness, 215 Main St., Davenport.
Secretary-Treasurer: Dr. W. F. Boiler, 105 East Iowa Ave., Iowa City.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John C. Brown, 350 St. Peter St., St. Paul.
Secretary-Treasurer: Dr. Hendrie W. Grant, Lowry Bldg., St. Paul.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Time: Semi-annually.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. M. Hendershott, 193, 11th St.. Portland.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Erza Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Fletcher D. Woodward, Box 162, University.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. S. S. Hall, Fairmont.
Secretary: Dr. G. A. Smith, Montgomery.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John R. Childs, 45 Edgewood Ave., Atlanta.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. William R. McKenzie, 11 E. Chase St., Baltimore.
Secretary: Dr. James J. Chisolm, Medical Arts Bldg., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. George J. Dennis, 25 E. Washnigton St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Edward D. King, 707 Race St., Cincinnati.
President-Elect: Dr. M. F. McCarthy, Union Central Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. William V. Mullin, 9204 Euclid Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 2064 E. 9th St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

Chairman: Dr. Ralph Butler, 2007 Chestnut St., Philadelphia.
Clerk: Dr. Karl M. Houser, 1826 Pine St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. M. Sellers, 717 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. Mood Knowles, 717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DETROIT OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Secretary-Treasurer: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. J. Richardson, 606 Penn St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robinson, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. A. E. Eubank, Arygle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

LOS ANGELES COUNTY MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Eugene R. Lewis, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of of each month, May to November, inclusive.

Medical Society of the County of Kings, Section on Otolaryngology

Chairman: Dr. John M. Auwerda, 878 President St., Brooklyn.
Secretary-Treasurer: Dr. Charles R. Weeth, 37 Remsen St., Brooklyn.
Place: Library Bldg., County Society, 1313 Bedford St., Brooklyn.
Time: 9 p. m., second Wednesday of January, March, May and November.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W.: Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: H. C. Smith, Medical Arts Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. W. R. Buffington, 211 Camp St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. John M. Loré, 240 West End Ave., New York.
Secretary: Dr. Robert E. Buckley, 2 E. 103d St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. James Homer McCready, 121 University Place, Pittsburgh.
Secretary-Treasurer: Dr. Roy C. Cooper, 510 S. Aiken Ave., Pittsburgh.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Emanuel U. Wallerstein, Professional Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. George E. Hourn, 607 N. Grand Blvd., St. Louis.
Secretary: Dr. Arthur M. Alden, Frisco Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. T. J. Walthall, 728 Main St., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Wilber F. Swett, 693 Sutter St., San Francisco.
Secretary: Dr. Robert Carson Martin, 384 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. Paul A. Remington, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubinstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

INDEX TO VOLUME 13

The asterisk (*) preceding the page number indicates an original article in the Archives. Subject entries are made for all articles. Author entries are made for original articles and for articles in society transactions. Book Reviews, Obituaries, and Society Transactions are indexed under these headings in their alphabetical order under the letters, B, O, and S, respectively.

PAGE

Abnormalities; agenesis of ear and congenital cyanosis ... 626
congenital, originating from second branchial arch ... 756
maldevelopments of auricle, external acoustic meatus and middle ear; microtia and congenital meatal atresia ... *1
of membraneous labyrinth ... 480
rare congenital deformities ... 753
Abscess, conjugate deviation of head and eyes, its value in diagnosis and localization of abscess of brain ... *683
lung, treatment with intratracheal injections ... 760
of brain, clinical and pathologic signs in ... 481
of brain; pathways of infection in abscess of brain; report of 5 cases ... *532
of the neck ... 770
orbital ... 636
orbital, following disease of sinus ... 302
otogenous, of brain, 9 cases ... 295
spontaneous cure in perforation of esophagus and mediastinal abscess ... 478
Actinomycosis of esophagus ... 773
Adenoid, adenoidectomy under local anesthesia ... *834
and tonsils (Progress in otolaryngology) ... *867
disturbances of handwriting in subjects with adenoid vegetations ... 632
Agranulocytosis: See Angina, agranulocytic
Aiken, T. G.: Compound fracture of malar bone with involvement of maxillary sinus ... 305
Albrecht: Injuries of ear at birth ... 477
Pathogenesis of cholesteatoma of middle ear ... 148
Alexander: Contribution to physiology of hearing ... 475
Allergy: See Anaphylaxis and Allergy
Amano, K. W.: Specific prophylaxis of streptococcic and pneumococcic meningitis ... 299
Amygdalectomy: See Tonsillectomy
Analgesia: See Anesthesia
Anaphylaxis and Allergy: anaphylactic changes in nasal mucosa of guinea-pigs ... *842
Anesthesia, local anesthesia for radical maxillary operation; an improved technic of administration ... *379
vestibular nystagmus in mixed nitrous oxide and oxygen anesthesia ... 296
Angina; agranulocytotica ... 891
agranulocytosis, malignant neutropenia; report of a case with apparent recovery ... *864
with atypical lymphocytemia in childhood ... 470
with lymphatic reaction in infancy ... 469
Anomaly: See Abnormality
Anson, B. J.: Sphenoidal sinusitis as a cause of optic neuritis; anatomy; relation of orbit and its content to paranasal sinuses ... 762
Antroscope, modified combined antroscope and irrigating cannula ... *275
Antrum, chronic maxillary sinusitis; analysis of 192 cases that came to operation ... *506
compound fracture of malar bone with involvement of maxillary sinus ... 305
cyst of antrums ... 635
local anesthesia for radical maxillary operation; an improved technic of administration ... *379
method of surgical intervention on antrum of Highmore ... 297
operative treatment of acute and chronic suppurations of maxillary sinus ... 757

Antrum—Continued PAGE
trephine for making a preturbinal opening in maxillary sinus ... *443
tuberculous-like lesions of antrum of Highmore induced by a foreign body ... 138
Aphasia, case of ataxic aphasia with paralysis of arm following otitic sinus thrombosis ... 465
Apparatus: See Instruments
Atelectasis: See Lung, collapse
Audiometer, practical value of ... 903
Auditory Organ: See Ear

Barbey, E.: Tonsils and reticulo-endothelial system ... 144
Barnes, H. A.: Fibrosarcoma of pharynx ... 772
Mucocele of frontal sinus ... 771
Beck: Displacement and kinking of esophagus ... 477
Extraordinary behavior of pulse in diseases of labyrinth and after radical operation ... 480
Belinoff: Treatment for corrosive acute and chronic esophagitis ... 479
Benjamins, C. E.: Experiments with substances inducing hay-fever ... 142
Berggren, S.: Cerebral changes in case of aural vertigo ... 144
Blood: See also Erythrocytes; Leukocytes
calcium, phosphorus and cholesterol in otosclerosis ... *77
prognostic value of examination of blood in otology ... *647
Boies, L. R.: Hunt's disease ... 639
Immature white blood cells in otologic infections ... *238
Bone conduction ... *489
conduction; mechanics of bone conduction as shown by a model ... 474
conduction; new test for borrowed bone conduction ... *822
rare diseases of bone of labyrinthine capsule ... 149

Book Reviews:
Atlas de anatomia del organo del oido; P. Belou ... 908
Tumors del Nasofarinx; A. E. Gay ... 307
Brain, abscess, clinical and pathologic signs in ... 481
abscess; conjugate deviation of head and eyes, its value in diagnosis and localization of abscess of brain ... *683
abscess, otogenous, 9 cases ... 295
abscess; pathways of infection in abscess of brain; report of 5 cases ... *532
concussion, neuro-otological observations in ... *785
tumor of brain and trauma ... 760
tumor; peculiar anomaly (pneumocele) of sphenoid bone in 2 cases of tumor of cerebral base ... 137
tumors, translabyrinthine operation on tumors of cerebellopontile angle ... 754
Breathing, tests of respiratory function ... 142
Bronchoscopy; bronchoscopic observations on obstructive pulmonary atelectasis ... *315
in diagnosis ... *445
in treatment ... *451
Bronchus, brass paper fastener in ... 473
Brunner, H.: Pathologic and clinical signs in central hearing tracts ... 477
Microscopic changes after a remote radical operation on ear ... 149
Brunschwig, A.: Subcutaneous mixed tumor of face not associated with salivary glands ... *52
Buckingham, T. W.: Herpes zoster oticus; report of case with facial paralysis ... *59
Bulbus Operation: See under Lateral Sinus
Bunch, C. C.: Age variations in auditory acuity ... *170
Race and sex variations in auditory acuity ... *423
Bursa Pharyngea: See under Pharynx

PAGE

Cahill, H. P.: Bilateral mastoiditis with sinus thrombosis..... 635
Labyrinthitis with facial paralysis..... 635
Cancer: See also Sarcoma; Tumor; etc.
carcinoma (slides)..... 635
differential diagnosis between foreign body and carcinoma of esophagus..... 478
endoscopic treatment of carcinoma of esophagus with diathermy..... 478
growth of cervical cancer..... 147
operation for carcinoma of pharynx..... 148
radium and surgery in cancer of tongue.. 472
radium treatment of buccal carcinoma... 472
radium treatment of cancer of tongue.... 472
Cannula, new modified combined antroscope and irrigating cannula.....*275
Carbon arclight, artificial sunlight in treatment of laryngeal tuberculosis; modified carbon arclight.....*326
Catarrh, dry mucosal catarrh and pharyngitis sicca..... 757
Cellulitis of face..... 770
Cerebrospinal fluid, discharge of, from external auditory canal.....*439
rhinorrhea, report of case of 18 years' duration *84
Chamberlin, W. B.: Delayed healing following simple operation on mastoid.....*829
Cholesteatoma, histologic study of formation of..... 467
of middle ear, diagnosis of..... 148
of middle ear, pathogenesis of..... 148
some new principles in its treatment, including test for its chemical demonstration *709
Chondromata of larynx..... 305
Cleminson, F.: Reflexes connected with the semicircular canals..... 149
Clerf, L. H.: Peroral endoscopy.....*444
Cochlea: See Labyrinth
Cold, etiology of common cold..... 473
Colorado Congress of Ophthalmology and Oto-Laryngology..... 885
Comer, M. C.: Cavernous sinus thrombosis in a child following tonsillectomy.....*733
Concussion of brain, neuro-otological observations in.....*785
Cork as plastic material for the correction of saddle noses..... 891
Cranium, fracture of base of skull..... 634
roentgen diagnosis of disease at base of skull *63
Craver, L. F.: Tumors of larynx and thyroid, roentgenographic study.....*658
Cyst of antrums..... 635

Dahmann, H.: Physiology of hearing; experimental investigation of mechanics of ossicular chain, and its behavior under tone and air pressure impulses.. 474
Daily, L.: Unusual case of 2 foreign bodies in lung.....*283
Davies, H. F.: Paralysis of vocal cords....*435
Davies, J. R., Jr.: Orbital abscess following disease of sinus..... 302
Deaf Mutes; deaf-mutism and education of deaf *98
speech and letter understanding, and its relation to hearing of speech in deaf-mutes 477
statistical report on cause of deaf-mutism in the Vymola Institute in Prague.... 890
use of space analyzer for examination of 290
Deafness: See also Hearing
hereditary, due to bilateral acoustic tumors 462
histologic observations on fenestrae in acquired deafness and comment on healing of surgical injury of stapes.... 888
new test for borrowed bone conduction...*822
spasm of auditory accomodative mechanism; functional basis of ear-drum retraction and of deafness for low tones *166
treatment of deaf infant..... 886
Deformities: See Abnormalities
Denker, A.: Faculty of hearing in fish..... 145
Despons: Vestibular disturbances accompanying epidemic encephalitis..... 150
Diathermy and high frequency currents in otorhinolaryngology 761
endoscopic treatment of carcinoma of esophagus with..... 478
Dickson, J. C.: Unusual case of 2 foreign bodies in lung.....*283

PAGE

Diphtheria carriers, tonsillectomy in..... 755
causes and prevention of mortality in.... 469
primary tracheobronchial.....*714
Dorrance, G. M.: So-called bursa pharyngea in man; its origin, relationship with adjoining nasopharyngeal structures and pathology.....*187
Treatment for stricture of oral pharynx.. 301
Dressing, use of paraffin oil for dressing wounds after operations on mastoid... 466
Druss, J. G.: Pathways of infection in abscess of brain; report of 3 cases....*532
Dyssynergia cerebellaris myoclonia (Hunt's disease) 639

Eagleton, W. P.: Unlocking of petrous pyramid for localized bulbar (pontile) meningitis secondary to suppuration of petrous apex; report of 4 cases with recovery in 3.....*386
Ear, agenesis of ear and congenital cyanosis 626
anatomy and physiology of ear; review of literature from October, 1929, to October, 1930.....*600
behavior of mucous membrane of middle ear following radical operation..... 749
birth injuries of..... 477
cholesteatoma of middle ear, diagnosis of 148
cholesteatoma of middle ear, pathogenesis of 148
deformities, case of rare congenital deformities 753
development of ear of Acanthias vulgaris 464
diagnostic errors in cases of otomycosis.. 752
discharge of spinal fluid from external auditory canal.....*439
Drum: See Tympanum
histologic observations on fenestrae in acquired deafness and comment on healing of surgical injury of stapes.... 888
infection; relation of infection of ear and infection of intestinal tract in infants 464
infections, immature white blood cells in otologic infections.....*238
inner: See Labyrinth
lymph channels connecting middle ear with cranial cavity..... 288
maldevelopments of auricle, external acoustic meatus and middle ear; microtia and congenital meatal atresia *1
microscopic changes after a remote radical operation on ear..... 149
Middle: See also Otitis Media
middle; functions of eustachian tube and its relation to chronic diseases of middle ear.....*676
middle, normal and pathologic physiology of 146
middle, pathogenesis of cholesteatoma of middle ear..... 148
muscles; function of intrinsic muscles of ear 475
physiology of intrinsic muscles of ear; investigations of mechanics of ossicular chain 475
practical significance of correct position of patient in otosurgical interventions in order to avoid secondary injuries.... 892
shock free methods of operation on..... 147
Edema of glottis..... 634
Electro-anesthetic currents of Araya in treatment of vertigo and tinnitus..... 287
Electrocoagulation, bipolar electrode for electrocoagulation of inferior turbinate.*442
Electrode, bipolar electrode for electrocoagulation of inferior turbinate.....*442
Emphysema of head and neck complicating tonsillectomy 468
Encephalitis, vestibular disturbances accompanying epidemic encephalitis 150
Endoscopy, peroral.....*444
Ephedrine, chronic effect of epinephrine and ephedrine on nasal mucosa..... *73
hydrochloride and epinephrine hydrochloride, after-effect of, on mucosa of nasal septum.....*255
Epinephrine: See Suprarenal Preparations
Epistaxis: See Nose, hemorrhage
Equilibrium, disturbances in, related to bilateral destruction of vestibular apparatus 289
Ersner, M. S.: Local anesthesia for radical maxillary operation; an improved technic of administration.....*379

PAGE

Ersner, W.: Local anesthesia for radical maxillary operation; an improved technic of administration....*379
Erythrocytes, sedimentation of erythrocytes in infections of middle ear..... 889
Esophagus, actinomycosis of..... 773
cancer, endoscopic treatment of, with diathermy 478
closure at mouth of esophagus after thoracoplasty for stenosis..... 478
corrosive acute and chronic esophagitis, treatment of..... 479
differential diagnosis between foreign body and carcinoma of esophagus..... 478
diseases *459
displacement and kinking of esophagus.. 477
perforation; spontaneous cure in perforation of esophagus and mediastinal abscess 478
recurrent esophageal pouch..... 638
stenosis of, congenital..... 148
stricture, prophylaxis of esophagus stricture following caustic accidents..... 478
swallowed silk thread as guide in esophageal instrumentation..... *94
ulcer of..... 773
Eustachian tube, functional examination of opening and closing of tuba auditiva, and some physiologic and pathologic observations 474
functions of, and relation to chronic diseases of middle ear.....*676
Exophthalmos, enormous mucocele of frontal sinus with exophthalmos and hyperostosis of orbital bone..... 631
Eyes, conjugate deviation of head and eyes, its value in diagnosis and localization of abscess of brain.....*683

Face, cellulitis of..... 770
plastic operation on..... 771
subcutaneous mixed tumor not associated with salivary glands..... *52
Fibrosarcoma of pharynx..... 772
Fischer: Hitherto unobserved anomaly of membranous labyrinth..... 480
Fistula; fistular symptoms induced by pressure on labyrinthine fistula..... 144
Foreign body, brass paper fastener in bronchus 473
case of tuberculous-like lesions of antrum of Highmore induced by a foreign body 138
differential diagnosis between foreign body and carcinoma of esophagus..... 478
in air and food passages.....*455
in lung..... 634
unusual case of 2 foreign bodies in lung..*283
Fowler, E. P.: Calcium, phosphorus and cholesterol in otosclerosis..... *77
Fox, N.: After-effect of epinephrine chloride and ephedrine hydrochloride on mucosa of nasal septum.....*255
Chronic effect of epinephrine and ephedrine on nasal mucosa..... *73
Fracture of base of skull..... 634
Fraser, J. S.: Maldevelopments of auricle, external acoustic meatus and middle ear; microtia and congenital meatal atresia *1
Frenzel: Differences in pitch between tine and stem tones in weighted "overtone-free" tuning forks, a source of error in Rinne test..... 475
Freund, E. M.: Paralysis of abducens nerve accompanying case of mastoidits, an unusual form of Gradenigo's syndrome..*717
Frey: Function of intrinsic muscles of ear.. 475
Friesner, I.: Pathways of infection in abscess of brain; report of 5 cases.....*532
Frontal Sinus, enormous mucocele of frontal sinus with exophthalmos and hyperostosis of orbital bone..... 631
mucocele of..... 771
mucocele of frontal sinus; its occurrence in a boy aged 13.....*597
osteomyelitis with frontal sinusitis; value of preserving anterior wall with attached periosteum.....*181

Gill, E. G.: Malignant neutropenia (agranulocytosis), report of case with apparent recovery *864
Glandular fever, Pfeiffer's..... 761

PAGE

Glioma, nasal, 2 cases..... 294
Glottis, edema of..... 634
Goldmann operation for diverticulum of hypopharynx 479
Goodale, R. L.: Cyst of antrums..... 635
Gouty deposits in pharyngeal mucosa..... 628
Gradenigo's syndrome, anatomic aspects.... *87
paralysis of abducens nerve accompanying case of mastoiditis; unusual form of Gradenigo's syndrome.....*717
with a consideration of "petrositis".....*347
correction to above article..... 596
Grahe, K.: Development of paranasal sinuses 142
Speech and letter understanding, and its relation to hearing of speech in deaf-mutes 477
Grünfelder's toe reflex..... 749
Guild, S. R.: Localization of tone receptors in human cochlea..... 146
von Gyergay: Functional examination of opening and closing of tuba auditiva, and some physiologic and pathologic observations 474

Hagens, E. W.: Otosclerosis, staining and chemical reactions.....*824
Hall, G. W.: Sphenoidal sinusitis as cause of optic neuritis; neurology: relation between involvement of sphenoid sinuses and optic neuritis..... 762
Halle: What does my intranasal operation on tear sac accomplish?..... 480
Handwriting, disturbances of handwriting in subjects with adenoid vegetations..... 632
Hansel, F. K.: Tonsils and adenoids.....*867
Hart, V. K.: Primary tracheobronchial diphtheria *714
Hastings, H.: Osteomyelitis associated with frontal sinusitis; value of preserving anterior wall with attached periosteum.*181
Hay fever, experiments with substances inducing hay-fever..... 142
Head, conjugate deviation of head and eyes, its value in diagnosis and localization of abscess of brain.....*683
Hearing: See also Deafness
age variations in auditory acuity.....*170
bone conduction.....*489
faculty of hearing in fish..... 145
for whisper and for conversational tone (contribution to question of tone hearing, speech understanding and forensics) 476
functional examination of hearing; deaf-mutism and education of deaf..... *98
fundamental tone of conduction apparatus and Rinne test..... 476
further observations on relation between endolymphatic pressure and hearing function 474
influence of outside noises on perception of sound..... 476
influence of very short acoustic waves on inner ear..... 476
localization of tone receptors in human cochlea 146
modified resonance theory for perception of ultramusical tones and of noises.... 749
new aid to hearing by use of bone conduction, principal bases of..... 476
new theory concerning mechanism of hearing 753
oculomotor reflexes provoked by sounds in persons with difficult hearing..... 467
pathologic and clinical signs in central hearing tracts..... 477
perception of ultramusical tones..... 146
physiology of hearing..... 475
physiology; experimental investigation of mechanics of ossicular chain, and its behavior under tone and air pressure impulses 474
practical value of audiometer..... 903
race and sex variations in auditory acuity.*423
sense of hearing survey of school children in Fergus Falls..... 464
speech and letter understanding, and its relation to hearing of speech in deaf-mutes 477
status of tests of hearing with a report of a speech interpretation test..... *47
Heermann: Seven years' experience with glass prosthesis of lower tear canal... 480
Spontaneous cure in perforation of esophagus and mediastinal abscess..... 478

PAGE

Herman, E. W.: Edema of glottis..... 634
Perichondritis of larynx..... 633
Recurrent esophageal pouch..... 638
Herpes zoster oticus, report of case with facial paralysis..... *59
Herzog: Mechanics of bone conduction as shown by a model..... 474
Hesse: Endoscopic treatment of carcinoma of esophagus with diathermy..... 478
Hewson, W.: Acute mastoiditis with basilar meningitis 303
High frequency currents and diathermy in otorhinolaryngology 761
Hirsch: Differential diagnosis between foreign body and carcinoma of esophagus.. 478
Influence of outside noises on perception of sound..... 476
Hoffmann: Prophylaxis of esophageal stricture following caustic accidents..... 478
Huizinga, E.: Galvanic vestibular reactions. 151
Hunt's Disease: See Dyssynergia cerebellaris myoclonica
Hurd, L. M.: Bipolar electrode for electrocoagulation of inferior turbinate.....*442
Hypopharynx: See Pharynx
Hypophysis: See Pituitary Body

Instrument, bipolar electrode for electrocoagulation of inferior turbinate.....*442
combined tongue depressor and pillar retractors *733
for cosmetic operation on nose..... 294
laryngeal tubes for indirect intubation and dilatation..... *96
new system of lighting for nasopharyngoscope; new operating attachments for nasopharyngoscope; modified combined antroscope and irrigating cannula.....*275
new type of tracheotomy tube with valve. 298
trephine for making a preturbinal opening in maxillary sinus.....*443
Insulin, use of insulin in postoperative mastoiditis with prolonged drainage.... 886
Iodized oil, method of outlining mucocele by injection of.....*836
sesame oil, method and results of injections of, for roentgenography of accessory cavities..... 758
Ivory, implantation of, in ozena, approved technic *726

Jacques, P.: Normal and pathologic physiology of middle ear..... 146
Jones, I. H.: Bone conduction.....*489
Josephson, E. M.: Spasm of auditory accommodative mechanism; functional basis of ear-drum retraction and of deafness for low tones.....*166

Kazanjian, V. H.: Plastic operation on face 771
Kelemen, G.: Action of tonsillary substance on growth of embryonal tissue in vitro. 143
Closure at mouth of esophagus after thoracoplasty for stenosis..... 478
Kelly, B.: Congenital stenosis of esophagus 148
Kemler, J. I.: Implantation of ivory in ozena, approved technic.....*726
Kerekes: Relation between endolymphatic pressure and hearing function..... 474
Kinney, C. E.: Mucocele of frontal sinus; its occurrence in a boy aged 13.....*597
Kirch, W.: Tumors of nose and throat.....*735
Kistner, F. B.: Histopathology and bacteriology of sinusitis with comments on postoperative repair.....*225
de Kleijn, A.: Reflex arc of vestibular nystagmus 150
Knudsen, V. O.: Bone conduction.....*489
Kobrak: Physiology of intrinsic muscles of ear; investigations of mechanics of ossicular chain..... 475
Kofler: Relations of diseases of tear sac to nose, and their treatment..... 479
Kolmer, J. A.: Present status of intracarotid method of treatment for pneumococcic and streptococcic meningitis..... 298
Specific prophylaxis of pneumococcic and streptococcic meningitis..... 299
Krainz: Fundamental tone of conduction apparatus and Rinne test..... 476

PAGE

Labyrinth, anomaly of membranous labyrinth hitherto unobserved..... 480
diseases of bone of labyrinthine capsule, rare 149
disturbances in equilibrium related to bilateral destruction of vestibular apparatus 289
extraordinary behavior of pulse in diseases of labyrinth and after radical operation 480
fistular symptoms induced by pressure on labyrinthine fistula..... 144
galvanic vestibular reactions..... 151
histologic changes in congenital osteogenesis imperfecta of membranous labyrinth 134
hydrostatic property of labyrinth and function of ductus endolymphaticus in fish 626
injuries; pathogenesis and clinical aspects of traumatic injuries of internal ear... 887
localization of tone receptors in human cochlea 146
neoformation of bone in labyrinthine cavity in case of destruction of labyrinth and meningitis..... 626
reflexes connected with semicircular canals 149
vestibular disturbances accompanying epidemic encephalitis..... 150
Labyrinthitis, cases complicating acute otitis media 751
rare anatomic features in cases of inflammation of labyrinth with bilateral sinus thrombosis 133
spontaneous nystagmus and inflammation of labyrinth..... 752
with facial paralysis..... 635
Lacrimal Organs, relations of diseases of tear sac to nose, and their treatment.. 479
results of endonasal operations on tear sac (examined functionally by x-rays). 479
secondary Toti-Mosher operation..... 633
seven years' experience with glass prosthesis of lower tear canal..... 480
what does my intranasal operation on tear sac accomplish? (Halle)..... 480
Laryngitis, non-diphtheritic infectious laryngitis 470
Laryngoscopy, direct.....*459
Larynx, acute laryngeal obstruction..... 470
chondromata of..... 305
laryngeal tubes for indirect intubation and dilatation..... *96
normal interior laryngeal functions which block view of glottal lips..... 899
perichondritis of..... 633
phonetic surgery on upper respiratory and alimentary tracts; substitution of natural or of artificial voice..... 139
roentgenographic anatomy and physiology of larynx and adjacent structures..... 775
specific infectious rheumatism involving upper digestive tract and larynx..... 630
tuberculosis, artificial sunlight in, modified carbon arclight.....*326
tumors of larynx and thyroid, roentgenographic study.....*658
Lateral Sinus, thrombosis of..... 774
value of bulbus operation..... 481
Ledoux: Growth of cervical cancer..... 147
Leiri: Perception of ultramusical tones.... 146
Leshin, N.: Are tonsillar recurrences entirely due to faulty operative technic?. *37
Leukocytes, immature white blood cells in otologic infections.....*238
Lewy, A.: Sodium iodide in hyperesthetic rhinitis *97
Linthicum, F. H.: Neuro-otological observations in concussion of brain.....*785
Lukens, R. M.: Chondroma of larynx..... 305
Lund, R.: Fistular symptoms induced by pressure on labyrinthine fistula..... 144
Lung, abscesses, treatment with intratracheal injections..... 760
bronchoscopic observations on obstructive pulmonary atelectasis.....*315
collapse, bronchoscopic observations on obstructive pulmonary atelectasis.....*315
foreign body in..... 634
foreign body; unusual case of 2 foreign bodies in.....*283
Lymph channels connecting middle ear with cranial cavity..... 288
nodes, tonsillectomy in treatment of acute cervical adenitis in children..... 469
Lymphocytemia, atypical, with angina..... 470

PAGE

MacFarlan, D.: Status of tests of hearing with a report of a speech interpretation test *47
McLeod, A.: Carcinoma 635
Neurofibroma 773
Malar Bone: See Zygoma
Maliniak, J. W.: Rhinophyma; its treatment and complications *270
Maljutin, E.: Stroboscopic examination of vocal students 141
Martin, R. C.: Intratemporal suture of facial nerve *259
Mastoid operation, delayed healing following simple operation *829
paraffin oil for dressing wounds after operations on mastoid 466
persistence of fetal septum in mastoid process 627
radical mastoid operation *28
surgery, secondary plastic closure of antrotomy wounds 750
Mastoiditis, acute, with basilar meningitis .. 303
bilateral, with sinus thrombosis 635
hysterical, case of 286
insulin in postoperative mastoiditis with prolonged drainage 886
lateral sinus thrombosis complicating masked mastoiditis 286
paralysis of abducens nerve accompanying case of mastoiditis, an unusual form of Gradenigo's syndrome *717
roentgen diagnosis in 888
treatment for otitis and mastoiditis in infancy 626
Maxillary Sinus: See Antrum
Mayer, O.: Factors of bone neoformation in cases of otosclerosis 149
Meesmann: Relations of diseases of tear sac to nose, and their treatment 479
Meltzer, P. E.: Anatomy and physiology of ear; review of literature from October, 1929, to October, 1930 ..... *600
Gradenigo's syndrome; anatomic aspects. *87
Thrombosis of lateral sinus 774
Ménière's Disease: See Vertigo
Meningitis, basilar, with acute mastoiditis .. 303
experimental studies regarding pathologic process in 482
intracarotid method of treatment of pneumococcic and streptococcic meningitis .. 298
neoformation of bone in labyrinthine cavity in case of destruction of labyrinth and meningitis 626
octogenic, therapy of 754
specific prophylaxis of pneumococcic and streptococcic meningitis 299
unlocking of petrous pyramid for localized bulbar (pontile) meningitis secondary to suppuration of petrous apex; report of 4 cases with recovery in 3 ..... *386
Meyers, I. L.: Conjugate deviation of head and eyes, its value in diagnosis and localization of abscess of brain *683
Miller, J. W.: Artificial sunlight in treatment of laryngeal tuberculosis; modified carbon arc-light *326
Mouth, cancer, radium treatment of 472
Mucocele, enormous mucocele of frontal sinus with exophthalmos and hyperostosis of orbital bone 631
method of outlining mucocele by injection of iodized oil *836
of frontal sinus 771
of frontal sinus; occurrence in boy aged 13 ..... *597
Mucosa, basement membrane in mucosa of upper respiratory passages *556
Muelwert: Influence of very short acoustic waves on inner ear 476
Mundt, G. H.: Practical value of audiometer 903
Murphy, W. E.: Anaphylactic changes in nasal mucosa of guinea-pigs 842
Muscles, function of intrinsic muscles of ear 475
paralysis of posterior cricothyroid muscle. 149
physiology of intrinsic muscles of ear; investigations of mechanics of ossicular chain 475
Mycosis, diagnostic errors in cases of otomycosis 752
Myerson, M. C.: Laryngeal tubes for indirect intubation and dilation *96
Mygind, S. H.: Tinnitus aurium 150
Mysel, P.: Orbital abscess 636

PAGE

Nadoleczny: Hearing for whisper and for conversational tone (contribution to question of tone hearing, speech understanding and forensics) 476
Nager, F. R.: Rare diseases of bone of labyrinthine capsule 149
Naris: See under Nose
Nasopharyngoscope, new system of lighting and new operating attachments for nasopharyngoscope *275
Nasopharynx, operative procedure for making passable cicatrically occluded nasopharynx 890
tumors; early diagnosis and importance of roentgenologic examination 290
Neck, abscess of 770
value of roentgen diagnosis in diseases of neck and upper respiratory tract .. 776
Nelson, R. F.: Adenoidectomy under local anesthesia *834
Nephrosis, relation of sinusitis in children to *159
Nerve, facial, intratemporal suture of facial nerve *259
optic; new method of demonstrating relation between sphenoid sinus and optic nerve 766
paralysis of abducens nerve accompanying a case of mastoiditis; unusual form of Gradenigo's syndrome *717
tumors, hereditary deafness due to bilateral acoustic tumors 462
Nervous system, rôle of sympathicus and parasympathicus in etiology of atrophic rhinitis *265
Neumann: Clinical and pathologic signs in abscess of brain 481
Neuritis, optic, secondary to postnasal sinus disease 135
optic, and sphenoid sinusitis, ocular symptoms in 764
optic, and sphenoidal sinusitis; relation between involvement of sphenoid sinuses and optic neuritis 762
retrobulbar 139
retrobulbar optic neuritis originating in nasal sinuses; new method of demonstrating relation between sphenoid sinus and optic nerve *846
symposium on sphenoidal sinusitis as a cause of optic neuritis; anatomy: relation of orbit and its contents to para-.nasal sinuses 762
Neurofibroma 773
Neutropenia, Malignant: See Agranulocytosis
New, G. B.: Tumors of nose and throat .. *735
Nose, Accessory Sinuses: See under Sinus
anaphylaxis; anaphylactic changes in nasal mucosa of guinea-pigs *842
atresia of posterior nares, congenital ... *570
deformity; correction of nasal disfigurements by readjustment of framework and tissues *584
glioma, 2 cases 294
hemorrhage, familial (hereditary) epistaxis 630
instrument for cosmetic operations on nose 294
method of nasal packing following submucous resection of septum which allows free nasal respiration *732
mucosa; after-effect of epinephrine hydrochloride and ephedrine hydrochloride on mucosa of nasal septum *255
mucosa; chronic effect of epinephrine and ephedrine on nasal mucosa *73
saddle cork as plastic material for correction of saddle noses 891
shock-free methods of operation on 147
syndrome of nasal nerve 136
tumors of nose and throat *735
Nostrils: See Nares
Nystagmus on change of recumbent position 481
reflex arc of vestibular nystagmus 150
spontaneous, and inflammation of labyrinth 752
vertical 750
vestibular, in mixed nitrous oxide and oxygen anesthesia 296

PAGE

Odors, mechanism of perception of odors by hematogenic route..... 472
Ophthalmoplegia, case of otogenous total ophthalmoplegia (contralateral)..... 631
Oppikofer: Is the Goldmann operation for diverticulum of hypopharynx to be recommended? 479
Orbit, abscess 636
abscess following disease of sinus..... 302
sphenoidal sinusitis as a cause of optic neuritis; anatomy: relation of orbit and its contents to paranasal sinuses. 762
Osteogenesis, histologic changes in cases of congenital osteogenesis imperfecta of membranous labyrinth..... 134
Osteomyelitis associated with frontal sinusitis; value of preserving anterior wall with attached periosteum.....*181
Otitis Media: See also Gradenigo's syndrome
cases of labyrinthitis complicating acute otitis media 751
etiology of middle ear suppuration..... 465
middle ear infection; common forms and their management..... 465
sedimentation of erythrocytes in infections of middle ear..... 889
treatment for otitis and mastoiditis in infancy 626
Otolaryngologic societies, directory of..... .....152, 308, 483, 641, 779, 909
Otology, prognostic value of examination of blood in.....*647
Otosclerosis, calcium, phosphorus and cholesterol in..... *77
factors of bone neoformation in cases of 149
roentgenographic study of pneumatization of temporal bone in..... 467
staining and chemical reactions.....*824
Ozena: See Rhinitis, Atrophic

Pack, G. T.: Tumors of larynx and thyroid; a roentgenographic study.....*658
Palate, cleft, Schoenborn-Rosenthal operation, from standpoint of speech specialist 482
growth of cervical cancer..... 147
Pancoast, H. K.: Roentgenographic anatomy and physiology of larynx and adjacent structures 775
Paraffin oil, use of paraffin oil for dressing wounds after operations on mastoid 466
Paralysis, facial, report of case with herpes zoster oticus..... *59
facial; labyrinthitis with facial paralysis 635
of abducens nerve accompanying case of mastoiditis, unusual form of Gradenigo's syndrome *717
of posterior cricothyroid muscle..... 149
of vocal cords.....*435
Pearlman, S. J.: Are tonsillar recurrences entirely due to faulty operative technic? *37
Peeler, C. N.: Primary tracheobronchial diphtheria *714
Pendergrass, E. P.: Value of roentgen diagnosis in diseases of neck and upper respiratory tract..... 776
Perichondritis of larynx..... 633
Petrous Pyramid: See Temporal Bone
Pfeiffer's glandular fever..... 761
Pharyngitis sicca and dry mucosal catarrh. 757
Pharynx, cancer, operation for..... 148
fibrosarcoma of..... 772
gouty depositis in pharyngeal mucosa.. 628
is the Goldmann operation for diverticulum of hypopharynx to be recommended? 479
phonetic surgery of upper respiratory and alimentary tracts; substitution of natural or of artificial voice..... 139
so-called bursa pharyngea in man; its origin, relationship with adjoining nasopharyngeal structures and pathology *187
stricture of oral pharynx, treatment.... 301
Pitman, L. K.: Functions of eustachian tube, its relation to chronic diseases of middle ear.....*676

Pitman, L. K.—Continued PAGE
New system of lighting for nasopharyngoscope; new operating attachments for nasopharyngoscope; modified combined antroscope and irrigating cannula....*275
Pituitary body, use of transsphenoidal puncture of pituitary for diagnosis and treatment 136
Plum, F. A.: Cerebrospinal rhinorrhea; report of case of 18 years' duration.. *84
Plummer-Vincent's syndrome..... 771
Pneumocele, peculiar anomaly (pneumocele) of sphenoid bone in 2 cases of tumor of cerebral base..... 137
Pomeranz, R.: Roentgen diagnosis of disease at base of skull..... *63
Popper: Experimental studies regarding pathologic process in meningitis..... 482
Portmann, G.: Vestibular disturbances accompanying epidemic encephalitis.. 150
Posture, experiments on..... 151
Post-graduate course at University of Bordeaux 885
Pressman, J. J.: Local anesthesia for radical maxillary operation; an improved technic of adminstration.....*379
Profant, H. J.: Gradenigo's syndrome, with a consideration of "petrositis".*347
correction to above article..... 596
Pulse, extraordinary behavior of pulse in diseases of labyrinth and after radical operation 480
Pyemia following tonsillectomy..... 629
pathogenesis of pyemia following sore throat, with reference to 2 cases..... 629
Pyramidal apex, clinical importance of structure of..... 465

Quincy, J. E.: Fracture of base of skull 634
Quinine, experimental investigations with.. 480

Radium and surgery in cancer of tongue.. 472
treatment of buccal carcinoma..... 472
treatment of cancer of tongue..... 472
Raiford, T. S.: Race and sex variations in auditory acuity.....*423
Rand, C. W.: Neuro-otological observations in concussion of brain.....*785
Rebattu: Paralysis of posterior cricothyroid muscle 149
Reflex, aural; disturbance in equilibrium related to bilateral destruction of vestibular apparatus..... 289
Grünfelder's toe reflex..... 749
oculomotor reflexes provoked by sounds in persons with difficult hearing..... 467
reflexes connected with semicircular canals 149
Rejtö, A.: Cholesteatoma, some new principles in its treatment, including test for its chemical demonstration.....*709
Diagnosis of cholesteatoma of middle ear. 148
Respiration, tests of respiratory function... 142
Respiratory tract, basement membrane in mucosa of upper respiratory passages..*556
value of roentgen diagnosis in diseases of neck and upper respiratory tract..... 776
Reticulo-endothelial system and tonsils..... 144
Retrouvey, H.: Vestibular disturbances accompanying epidemic encephalitis..... 150
Rheumatism, specific infectious rheumatism involving upper digestive tract and larynx 630
specific toxins of bacteria isolated from tonsils of patients with articular rheumatism 135
Rhinitis, implantation of ivory in ozena, approved technic.....*726
results of surgical constriction induced for treatment of..... 293
rôle of sympathicus and parasympathicus in etiology of atrophic rhinitis.....*265
sodium iodide in hyperesthetic rhinitis... *97
vasomotor 471
Rhinophyma, its treatment and complications *270
Rhinoplasty: See under Nose
Rhinorrhea, cerebrospinal, report of case of 18 years' duration..... *84
Rhinosporidium seeberi; pathological histology and report of third case from United States..... 631
Rinne test, differences in pitch between tine and stem tones in weighted "overtone-free" tuning forks, a source of error in Rinne test..... 475

Rinne Test—Continued PAGE
fundamental tone of conduction apparatus and Rinne test..... 476
Roentgen Ray diagnosis in mastoiditis..... 888
diagnosis of disease at base of skull..... *63
method and results of injections of iodized sesame oil 40 per cent for roentgenography of accessory cavities..... 758
treatment of hypertrophied tonsils..... 628
value in diagnosis in disease of neck and upper respiratory tract..... 776
Roorda: Resonance of tones in sea shells... 147
Rosenberger, H. C.: Mucocele of frontal sinus; its occurrence in a boy aged 13. *597
Roth, E.: Discharge of cerebrospinal fluid from external auditory canal..... *439
Method of nasal packing following submucous resection of septum which allows free nasal respiration..... *732
Russell, G. O.: Normal interior laryngeal functions which block view of glottal lips 899
Ruttin: Nystagmus on change of recumbent position 481

Schall, L.: Secondary Toti-Mosher operation 633
Schenk: Reflex arc of vestibular nystagmus. 150
Schilling: Schoenborn-Rosenthal operation for cleft palate from standpoint of speech specialist..... 482
Schlander: Value of bulbus operation..... 481
Schmaltz: Experiments on posture..... 151
School children, sense of hearing survey of school children in Fergus Falls..... 464
Seiferth: Experimental investigations with quinine 480
Semicircular Canals: See Labyrinth
Sercer, A.: Tests of respiratory function... 142
Shambaugh, G. E., Jr.: Basement membrane in mucosa of upper respiratory passages *556
Plummer-Vincent's syndrome..... 771
Sheehan, J. E.: Correction of nasal disfigurements by readjustment of framework and tissues..... *584
Shells, resonance of tones in sea shells..... 147
Sinus: See also Frontal Sinus; Lateral Sinus; etc.
accessory nasal sinuses; method and results of injections of iodized sesame oil 40 per cent for roentgenography..... 758
accessory sinuses, results of endonasal operations on..... 291
analysis of 100 consecutive nasal sinus cases treated conservatively..... 471
histopathology and bacteriology of sinusitis with comments on postoperative repair *225
optic neuritis secondary to postnasal sinus disease 135
orbital abscess following disease of..... 302
paranasal, development of..... 142
paranasal sinus; method of outlining mucocele by injection of iodized oil... *836
physical therapy measures in selected cases of sinusitis..... 631
relation of sinusitis to nephrosis in children *159
retrobulbar optic neuritis originating in nasal sinuses; new method of demonstrating relation between sphenoid sinus and optic nerve..... *846
sphenoidal sinusitis as cause of optic neuritis; anatomy: relation of orbit and its contents to paranasal sinuses..... 762
Smell, mechanism of perception of odors by hematogenic route..... 472
Smith, J. M.: Radical mastoid operation... *28
Smyth, D. C.: Foreign body in lung..... 634
Ulcer of esophagus..... 773
Societies, Colorado Congress of Ophthalmology and Oto-Laryngology..... 885
local, directory of..154, 310, 485, 643, 781, 911
national, directory of.....
.....152, 308, 483, 641, 779, 909
sectional, directory of.....
.....152, 308, 483, 641, 779, 909
state, directory of..153, 309, 484, 642, 780, 910
Society Transactions:
American Society for Study of Disorders of Speech..... 893
Chicago Laryngological and Otological Society 762, 899

Society Transactions—Continued PAGE
College of Physicians of Philadelphia, Section on Otolaryngology.....298, 775
Collegium Oto-Rhino-Laryngologicum..... 141
German Otolaryngologic Society..... 474
Massachusetts Eye and Ear Infirmary, Oto-Rhino-Laryngological Services..633, 770
Sodium Iodide in hyperesthetic rhinitis..... *97
Sonnenschein, R.: Functional examination of hearing; deaf-mutism and education of deaf *98
Sonntag: Principal bases of a new aid to hearing by use of bone conduction..... 476
Spasm of auditory accomodative mechanism; functional basis of ear-drum retraction and of deafness for low tones..*166
Speech drills, value of, a symposium..... 897
is stuttering a neurotic or psychoneurotic manifestation? a symposium..... 894
normal interior laryngeal functions which block view of glottal lips..... 899
rôle of sinistrality and ambidexterity as causative factor in stuttering, a symposium 896
use of suggestion in attempting to induce better speech in stutterer, a symposium 898
Sphenoid Bone, peculiar anomaly (pneumocele) in cases of tumor of cerebral base 137
Sphenoid Sinus; new method of demonstrating relation between sphenoid sinus and optic nerve..... 766
symposium on sphenoidal sinusitis as cause of optic neuritis..... 762
symposium on sphenoid sinusitis as a cause of optic neuritis; anatomy: relation of orbit and its contents to paranasal sinuses..... 762
sphenoidal sinusitis and optic neuritis; ocular symptoms..... 764
sphenoidal sinusitis as cause of optic neuritis; neurology: relation between involvement of sphenoid sinuses and optic neuritis 762
Spies: Question of narcosis for tonsillectomy 482
Staining, otosclerosis; staining and chemical reactions..... *824
Stammering: See under Speech
Stevenson, W.: Chronic maxillary sinusitis; analysis of 192 cases that came to operation *506
Stewart, J. P.: Congenital atresia of posterior nares..... *570
Streptococcus, sporadic septic sore throat due to streptococcus epidemicus..... 469
Stroboscopic examination of vocal students. 141
Stuttering: See under Speech
Suker, G. F.: Sphenoidal sinusitis and optic neuritis; ocular symptoms..... 764
Sunlight, artificial sunlight in treatment of laryngeal tuberculosis; modified carbon arclight *326
Suprarenal Preparations; chronic effect of epinephrine and ephedrine on nasal mucosa *73
epinephrine hydrochloride and ephedrine hydrochloride, after-effect of, on mucosa of nasal septum..... *255
Surgery, practical significance of correct position of patient in otosurgical interventions in order to avoid secondary injuries 892

Temporal Bone, clinical aspects of inflammatory disease of apex of petrous portion of..... 886
Gradenigo's syndrome with a consideration of "petrositis"..... *347
correction 596
roentgenographic study of pneumatization of temporal bone in otosclerosis..... 467
suppuration of; pathology, symptomatology and surgical treatment..... 462
unlocking of petrous pyramid for localized bulbar (pontile) meningitis secondary to suppuration of petrous apex; report of 4 cases with recovery in 3..... *386
Tetanus, auricular, and its relation to ocular tetanus..... 286
Thoracoplasty, closure at mouth of esophagus after thoracoplasty for stenosis 478
Thorburn, T. R.: Combined tongue depressor and pillar retractors..... *733

PAGE

Thread, swallowed silk thread as guide in esophageal instrumentation..... *94
Throat, sore..... 629
sore; pathogenesis of pyemia following sore throat, with reference to 2 cases.. 629
sporadic septic sore throat due to streptococcus epidemicus..... 469
tumors of nose and throat.....*735
Thrombosis, bilateral mastoiditis with sinus thrombosis 635
case of ataxic aphasia with paralysis of arm following otitic sinus thrombosis.. 465
cavernous sinus thrombosis in a child following tonsillectomy.....*733
lateral sinus thrombosis complicating masked mastoiditis..... 286
method to prove absence of obturating sinus thrombosis or jugularis thrombosis in a case with a pyemic fever curve 889
of lateral sinus..... 774
ophthalmologic aspects of cranial sinus thrombosis 759
rare anatomic features in cases of inflammation of labyrinth with bilateral sinus thrombosis..... 133
Thyroid, tumors of larynx and thyroid, roentgenographic study.....*658
Tinnitus aurium..... 150
treatment of vertigo and tinnitus by electro-anesthetic currents of Araya... 287
Tobey, H. G.: Abscess of neck..... 770
Cellulitis of face..... 770
Toe reflex, Grünfelder's..... 749
Tongue, cancer, radium and surgery in.... 472
cancer, radium treatment of..... 472
combined tongue depressor and pillar retractors *733
Tonsillectomy, are tonsillar recurrences entirely due to faulty operative technic? *37
aspiration in tonsillectomy; comparative merits of posture and other factors; bronchoscopic study of 110 patients.. 627
by Ballenger-Sluder method, the operation of choice in children..... 469
cavernous sinus thrombosis in child following *733
combined tongue depressor and pillar retractors *733
during or shortly after an acute infection of tonsils..... 291
emphysema of head and neck complicating tonsillectomy 468
hemorrhage after, arrest of; reply to an article by Gumperz..... 891
in diphtheria carriers..... 755
in acute cervical adenitis..... 469
pyemia following tonsillectomy..... 629
question of narcosis for..... 482
results of..... 468
Tonsils, action of tonsillary substance on growth of embryonal tissue in vitro.... 143
and adenoids (Progress in otolaryngology) *867
and reticulo-endothelial system..... 144
changes in leukocytal blood picture after experimental irritation of tonsils..... 143
congenital anomalies originating from second branchial arch..... 756
roentgen treatment of hypertrophied tonsils 628
specific toxins of bacteria isolated from tonsils of patients with articular rheumatism 135
sudden death from hyperplasia of lingual tonsil 755
tuberculosis of, primary and secondary.. 756
Tracheobronchial diphtheria, primary.....*714
Tracheotomy, indications, technic and accidents of..... 473
modification in..... 759
tube, new type, with valve..... 298
Tremble, G. E.: Method of outlining mucocele by injection of iodized oil.....*836
Trephine for making a preturbinal opening in maxillary sinus.....*443
Tuberculosis, artificial sunlight in treatment of laryngeal tuberculosis; modified carbon arclight.....*326

Tuberculosis—Continued PAGE
primary and secondary tuberculosis of tonsils 756
Tucker, G.: Bronchoscopic observations on obstructive pulmonary atelectasis...,*315
New type of tracheotomy tube with valve 298
Tumors, hereditary deafness due to bilateral acoustic tumors..... 462
of brain and trauma..... 760
of cerebellopontile angle, translabyrinthine operations on..... 754
of cerebral base, peculiar anomaly (pneumocele) of sphenoid bone in 2 cases of. 137
of larynx and thyroid, roentgenographic study *658
of nose and throat.....*735
of rhinopharynx with cranial propagation, early diagnosis and roentgenologic examination 290
subcutaneous mixed tumor of face not associated with salivary glands..... *52
Tuning forks, differences in pitch between tine and stem tones in weighted "overtone-free" tuning forks, a source of error in Rinne test..... 475
Turbinate, bipolar electrode for electrocoagulation of inferior turbinate.....*442
Tympanic pyramid, suppuration at tip of... 151
Tympanum, spasm of auditory accommodative mechanism; functional basis of ear-drum retraction and of deafness for low tones.....*166

Ulcer of esophagus..... 773
Underwood, H. L.: Trephine for making preturbinal opening in maxillary sinus.*443
Unterberger: Results of endonasal operations on tear sac (examined functionally by x-rays)..... 479

Vail, H. H.: New method of demonstrating relation between sphenoid sinus and optic nerve..... 766
Retrobulbar optic neuritis originating in nasal sinuses; new method of demonstrating relation between sphenoidal sinus and optic nerve.....*846
Vertigo, cerebral changes in case of aural vertigo 144
Ménière's syndrome..... 751
neuro-otological observations in concussion of brain.....*785
treatment by electro-anesthetic currents of Araya..... 287
Vestibule: See Labyrinth
Vinson, P. P.: Swallowed silk thread as a guide in esophageal instrumentation... *94
Vocal cords, paralysis of.....*435
stroboscopic examination of vocal students 141
Voice, phonetic surgery on upper respiratory and alimentary tracts; substitution of natural or of artificial voice.... 139
Voss, O.: Suppuration at tip of tympanic pyramid 151

Wachsberger, A.: Rôle of sympathicus and parasympathicus in etiology of atrophic rhinitis *265
Weille, F. L.: Actinomycosis of esophagus. 773
Weiss, A.: Prognostic value of examination of blood in otology.....*647
Wieder, H. S.: New test for borrowed bone conduction *822
Wimmer, S. D.: Relation of sinusitis to nephrosis in children.....*159
Wiskovsky, B.: Changes in leukocytal blood picture after experimental irritation of tonsils 143
Wojatschek, W.: Shock-free methods of operation on ear and nose..... 147

Zange, J.: Operation for carcinoma of pharynx 148
Zwaardemaker: Resonance of tones in sea shells 147
Zygoma, compound fracture of malar bone with involvement of maxillary sinus... 305

VOLUME 13 NUMBER 6

ARCHIVES OF OTOLARYNGOLOGY

EDITORIAL BOARD

GEORGE E. SHAMBAUGH, Chicago
ISIDOR FRIESNER, New York
CHEVALIER JACKSON, Philadelphia
RALPH A. FENTON, Portland, Ore.
ROBERT C. LYNCH, New Orleans
GEORGE FETTEROLF, Philadelphia

JUNE, 1931

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $6.00

Entered as Second-Class Matter, January 14, 1925, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879. Acceptance for mailing at special rate provided for in Section 1103, Act of October 3, 1917, authorized January 22, 1925.

Copyright, 1931, by the American Medical Association

THE ARCHIVES OF OTOLARYNGOLOGY is published by the American Medical Association to stimulate research in the field of laryngology and otology, and to disseminate knowledge in this department of medicine.

Manuscripts for publication, books for review, and correspondence relating to the editorial management should be sent to Dr. George E. Shambaugh, Editor-in-Chief, 122 South Michigan Avenue, Chicago, or to any other member of the Editorial board. Communications regarding subscriptions, reprints, etc., should be addressed, ARCHIVES OF OTOLARYNGOLOGY, American Medical Association, 535 North Dearborn Street, Chicago.

Articles are accepted for publication on condition that they are contributed solely to the ARCHIVES OF OTOLARYNGOLOGY. Manuscripts must be typewritten, preferably double spaced, and the original copy should be submitted. Zinc etchings and halftones of illustrations will be supplied by the publisher when the original illustrations warrant.

Footnotes and bibliographies should conform to the style of the Quarterly Cumulative Index Medicus, published by the American Medical Association. This requires, in order given: name of author, title of article, name of periodical, with volume, page, month—day of month if weekly—and year. A complete list of abbreviations for standard periodicals, together with a full discussion of the style of the A. M. A. publications, appears in The Art and Practice of Medical Writing, a comprehensive book on the preparation of medical manuscripts, published by the American Medical Association. Price, $1.50.

Matter appearing in the ARCHIVES OF OTOLARYNGOLOGY is covered by copyright, but, as a rule, no objection will be made to its reproduction in reputable medical journals if proper credit is given. However, the reproduction for commercial purposes of articles appearing in the ARCHIVES OF OTOLARYNGOLOGY, or in any of the other publications issued by the Association, will not be permitted.

Authors will receive one hundred reprints free; additional reprints may be obtained at cost.

The ARCHIVES OF OTOLARYNGOLOGY is issued monthly. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.40; foreign, $6.75, including postage. Single copies, 75 cents, postpaid.

Checks, etc., should be made payable to the AMERICAN MEDICAL ASSOCIATION.

OTHER PERIODICAL PUBLICATIONS
of the American Medical Association

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (two volumes): Domestic, $7.00; Canadian, $8.50; foreign, $9.50. Single copies, 25 cents.

ARCHIVES OF INTERNAL MEDICINE—Monthly. Devoted to the publication of advanced, original clinical and laboratory investigations in internal medicine. Illustrated. Annual subscription price (two volumes): Domestic, $5.00; Canadian, $5.40; foreign, $5.75. Single copies, 75 cents.

AMERICAN JOURNAL OF DISEASES OF CHILDREN—Monthly. Presents pediatrics as a medical science and as a social problem. It includes carefully prepared collective abstracts based on recent pediatric literature, abstracts from foreign and domestic literature, book reviews, society transactions, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY—Monthly. Devoted to advancing the knowledge of and progress in cutaneous diseases and syphilis. Publishes original contributions and full abstracts of the literature on these two subjects, transactions of the important dermatological societies, book reviews, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation of original articles on nervous and mental diseases, with abstracts from foreign and domestic literature, book reviews, society transactions, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF SURGERY—Monthly. Devoted largely to the investigative and clinical phases of surgery, with monthly reviews on orthopedic and urologic surgery. Well illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original contributions in the field of pathology, with abstracts from foreign and domestic literature, book reviews, the transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.40; foreign, $6.75. Single copies, 75 cents.

ARCHIVES OF OPHTHALMOLOGY—Monthly. Includes original articles on diseases of the eye, abstracts from foreign and domestic literature, book reviews, transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $8.00; Canadian, $8.40; foreign, $8.75. Single copies, 85 cents.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and author index to the worth-while current medical literature of the world. Issued four times a year. Second and fourth volumes bound for permanent reference. Subscription price, calendar year: Domestic, $12.00; Canadian, $12.50; foreign, $12.75.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn St. CHICAGO

CONTENTS

PAGE

Neuro-Otological Observations in Concussion of the Brain. Fred H. Linthicum, M.D., and Carl W. Rand, M.D., Los Angeles 785

A New Test for Borrowed Bone Conduction. Henry S. Wieder, M.D., Philadelphia 822

Otosclerosis: Staining and Chemical Reactions. E. W. Hagens, M.D., Chicago 824

Delayed Healing Following the Simple Operation on the Mastoid. William B. Chamberlin, M.D., Cleveland 829

Adenoidectomy Under Local Anesthesia. Roy F. Nelson, M.D., Oakland, Calif 834

Method of Outlining Mucocele by the Injection of Iodized Oil. G. Edward Tremble, M.D., Montreal, Canada 836

Anaphylactic Changes in the Nasal Mucosa of Guinea-Pigs. William E. Murphy, M.D., Boston 842

Retrobulbar Optic Neuritis Originating in the Nasal Sinuses: A New Method of Demonstrating the Relation Between the Sphenoid Sinus and the Optic Nerve. Harris H. Vail, M.D., Cincinnati 846

Clinical Notes:

Malignant Neutropenia (Agranulocytosis): Report of a Case with Apparent Recovery. E. G. Gill, M.D., Roanoke, Va 864

Progress in Otolaryngology. A Summary of the Bibliographic Material Available in the Field of Otolaryngology: Tonsils and Adenoids. French K. Hansel, M.D., St. Louis 867

News and Comment 885

Abstracts from Current Literature 886

Society Transactions:

American Society for the Study of Disorders of Speech .. 893

Chicago Laryngological and Otological Society 899

Book Reviews 908

Directory of Otolaryngologic Societies 909

General Index 915